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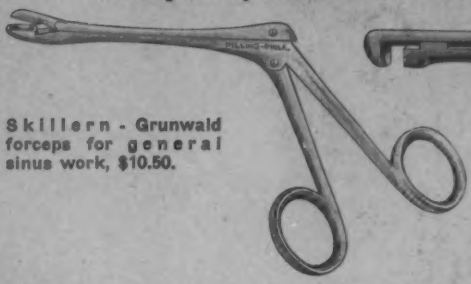

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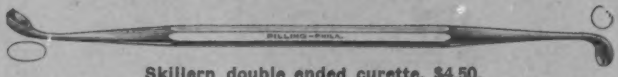

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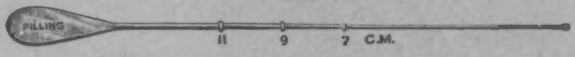

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THE
LARYNGOSCOPE.

VOL. XL MAY, 1930. No. 5

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

SYMPOSIUM: BRAIN ABSCESS.

BRAIN ABSCESS FROM THE STANDPOINT OF THE OPHTHALMOLOGIST.*†

DR. WM. L. BENEDICT, Rochester.

Rarely, if ever, does the ophthalmologist, in the capacity of a primary consultant, see a case of abscess of the brain. He rather is called on to interpret possible ocular phenomena in order to corroborate the diagnosis so far as these phenomena are determinate. The diagnosis of abscess of the brain is difficult even with ocular signs and symptoms, none of which is pathognomonic; all occur with other lesions of the brain, such as tumor of the brain, meningitis and encephalitis. There are also to be considered extradural abscess, osteomyelitis of the skull and tumor of the dura with softening or hemorrhage; these are conditions which may occur concomitant with diseases of the sinuses and the mastoid and give rise to ocular signs similar to those which are the result of abscess of the brain; they complicate the diagnosis. Although there is marked variability in the frequency with which ocular signs have been noted in cases of abscess of the brain, as reported in the literature, it is recognized quite generally that the absence of any ocular signs or symptoms makes the diagnosis less certain. It may be that the occurrence of ocular signs is reported as being more common by those who have

*Read before Amer. Acad. of Ophth. and Oto-Laryn., Atlantic City, Oct. 22, 1929.

†From the Dept. of Ophthalmology, The Mayo Clinic.

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access to ophthalmologic consultation, as some of the signs are elicited with difficulty or are evanescent in character and discovered only by repeated examination. In this country, ophthalmology is combined with otology by a great majority of those engaging in the practice of these specialties. For the members of this Academy, therefore, it is impossible to view from distant standpoints a series of phenomena occurring in a case of suspected abscess of the brain. It is possible to consider the significance of certain signs and symptoms of the visual organs and to correlate them with other data, but to base conclusions on ophthalmologic data alone would be sheer folly. Therefore ocular signs and symptoms must be considered in relation to all the data in a given case, the standpoint of the ophthalmologist being only a little aside from that of any other consultant on the case.

First to be considered, therefore, is the history of the onset and course of the pathologic process under consideration. Previous illnesses, accidents, operations, exposures and habits, as well as occupation, habitual colds, headaches and a host of other factors that might influence any symptom or sign or predispose the patient to intracranial disturbance must be carefully investigated and taken into consideration. The etiology of abscess of the brain is often obscure. As Kennedy stated, the diagnosis is fairly difficult in the absence of a direct lead from the etiology. The history of a discharging ear, previous mostoidectomy or a chronic disease of the paranasal sinus is often of primary significance in distinguishing abscess from other pathologic conditions and in determining the probable site of the lesion. It should also be borne in mind that multiple hematogenous abscesses may occur from a septic process elsewhere in the body, such as abscess of the lung or suppurative cystitis, while at the same time suppurative mastoiditis or disease of a sinus exists without direct relationship to the lesion of the brain.

One must also bear in mind the development of abscess of the brain, which was so well described by MacEwen in 1893. He divided the clinical sequence of events produced by the formation of an abscess of the brain into: *1.* The initiatory stage, characterized by severe pain in the head, vomiting, rigor and fever; *2.* the second stage, characterized by pain in the head (which can be elicited only by percussion), slow cerebration, want of sustained attention, apyrexia, slow pulse, paralysis and optic neuritis; and *3.* the terminal stage, in which there is either leakage of the pus into the membranes or ventricles, accompanied by fever, convulsions and coma, or complete encapsulation of the abscess, which may remain quiescent for

a long period and may finally become absorbed, in some cases without surgical interference, as illustrated in Case 1.

Case 1: A woman, age 52 years, was referred to the Mayo Clinic in August, 1928, because of left exophthalmos and diplopia. Her attention had been directed to a swelling and drooping of the left upper lid nine months previously and diplopia occurred two months later. During the seven months previous to her visit to the clinic the condition of the orbit had not changed noticeably. Four weeks before admission, a "diagnostic" opening had been made into the frontal sinus after the method of Halle. Mucus was not evacuated, even though the Roentgenogram showed marked pathologic change in the frontal region. Vision in the right eye was 6/5 and in the left eye 6/12; the fields and fundi were normal (see Fig. 1).

Fig. 1 (Case 1). Mucocele of left frontal sinus; displacement of the globe downward and exophthalmos; empyema of right frontal sinus was present.

An incision was made through the brow on the nasal side down to the margin of the orbit and a quantity of thick, sebaceous material escaped. The fascia was separated from the orbital margin, allowin gaccess into a fairly large frontal sinus, which was completely filled with thick, gray, paste-like material. The wall of a cyst was found and removed. The wound was closed without drainage as the nasofrontal duct was large enough to admit a 7 m.m. probe. There was no communication with the frontal sinus on the right side. The sinus was not deep and the roof was smooth and glistening. The floor was partly eroded, allowing for the pressure into the orbit causing the exophthalmos. The pathologist reported "hemorrhagic inflammatory tissue."

The patient was dismissed from the hospital on the fourth day. The following day the wound was opened, since the sinus evidently was not draining into the nose, and considerable pus came from the sinus. Drains were inserted into the wound, which continued to discharge pus. On the twelfth day after operation she complained of considerable headache. When she was lying on the left side or changing position from lying to sitting, the pain about the left side of the head was very severe. She had a chill and the temperature rose to 100.8° F. She was again sent to the hospital and the wound was opened wide for better drainage. Considerable pus was obtained from two walled-off pockets on the temporal side of the wound. The temperature varied from 98.6° to 100° until on the sixteenth day it rose from 98° to 101°. The patient became stuporous and drowsy, refused food, and complained only of headache. She died of respiratory failure, the heart continuing to beat for a few minutes after respiration ceased.

Necropsy disclosed a rather large brain abscess of the left frontal lobe with perforation of the orbital plate and localized meningitis. Although the sinuses were not connected, the right frontal sinus was filled with pus. The abscess in the left frontal lobe was connected with the left frontal sinus, and by another small opening into the lateral ventricle.

The original pathologic condition was mucocele of the left frontal sinus, and purulent infection of the right frontal sinus. The brain abscess was not walled off but consisted of a pocket of pus on a line of infection extending from the sinus to the ventricles. This illustrated MacEwen's third stage with leakage of pus into the ventricles, although it was accompanied by slight fever, without convulsion or coma. Surgical interference would have been of no avail for there were no localizing signs and the abscess was not encapsulated. This type is sometimes referred to as fulminating abscess of the brain.

Clinically, the ocular syndromes produced by encapsulated abscess of the brain are the same as those produced by tumor of the brain. When encapsulation has not taken place the process must be considered as one of inflammation or softening. The significance of this differentiation to the surgeon cannot be over-emphasized. In the Mayo Clinic it has been noted that the best results are obtained by operation in cases in which the opthalmologic and neurologic syndromes could not be distinguished from those produced by a tumor of the brain in the same situation. While the abscess is developing the choked disc fluctuates, becoming stationary when the abscess

becomes encapsulated. In cases in which operation is delayed until encapsulation has occurred, as shown by the disc, the postoperative course is less stormy and the mortality rate is lower. As a rule maximal choking occurs in a short time and surgical procedures may advisedly be deferred until the choked disc is stationary or has begun to recede. It is, therefore, in the second and third stages of abscess of the brain, as outlined by MacEwen, that the ocular phenomena are of the most diagnostic and localizing value. In other words, from the standpoint of the ophthalmologist, the signs and symptoms of abscess of the brain are those of tumor of the brain. They are of great significance when present, but their absence is not conclusive.

The most common ocular sign of abscess of the brain is choked disc, present in about a third of the cases. A choked disc may not occur in cases of abscess of the brain or tumor of the brain; this depends on the size and especially the site of the lesion. The absence of a choked disc consequently is not proof of the absence of abscess; it is known that a certain type of abscess, the extradural, is not as a rule accompanied by choked disc. Lillie and Lillie reported from the Mayo Clinic four cases of choked disc associated with surgical mastoiditis; three of the patients had parasinus abscess and one patient had pachymeningitis with a large cholesteatoma pressing on the sinus and cerebellum. All the patients recovered and the choked disc receded. The same principle which, on the whole, is generally admitted for neoplasms has an effect in the abscess. The marked increase of the cerebral volume can be well tolerated without choked disc if the site of the lesion does not impede the intracerebral circulation of the spinal fluid.

Certain abscesses are favorably situated to produce choked disc, such as those in the temporal lobe; others, such as some cerebellar tumors, rarely cause it. Abscess of the frontal lobe, according to the consensus of opinion in the literature, causes choked disc infrequently unless the abscess is very large, yet in 11 cases of definitely encapsulated abscess of the frontal lobe Lillie found bilateral choked discs ranging from one to six diopters, in eight. Abscess of the temporal lobe accompanied by choked disc is the most common, almost always arising from diseases of the ear. The degree of choked disc is also most frequently found to be greater, four to six diopters, in contrast to lesions of the cerebellum, in which the swelling rarely exceeds two diopters; often slight blurring of the margins of the disc is all that is found. Choked disc usually appears bilaterally and thus is of little localizing value. In many cases,

however, the papilledema is not equal and cases are reported in which the lesion was found to be on the side on which the edema was greater. In other cases, however, the lesion was found on the side on which the edema was less. The reason for this probably lies in the occurrence of papilledema on one side earlier than on the other, or increasing edema of one disc occurring simultaneously with decreasing edema of the other. It is well known that choked disc does not occur in atrophic discs and that the papilledema recedes in an eye after atrophy is once well under way. In cases of certain lesions of the frontal lobe, direct pressure on the optic nerve may produce atrophy with or without previous choking of the disc, the basal frontal syndrome of Kennedy. Only one abscess so situated has been observed at the Mayo Clinic.

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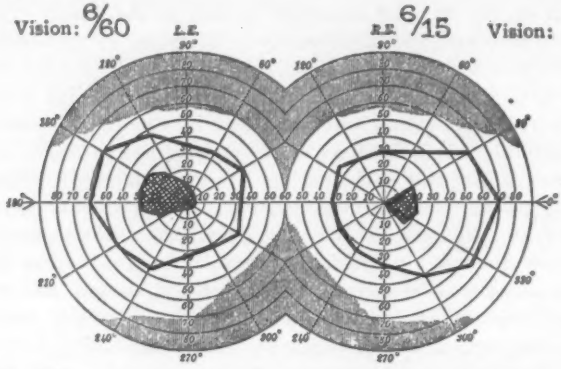


Fundi:

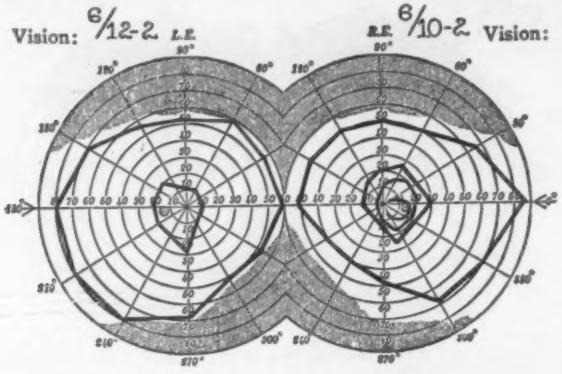
Fig. 2 (Case 2). Fields on patient's admission: concentric contraction of fields; enlargement of blind spots with cecocentral scotoma of the left eye.

Case 2: A boy, age 14 years, was brought to the Mayo Clinic, Oct. 27, 1926, because of headaches. Eight months previously he had had an ordinary cold in the head with fever. Eight days later the left eyelids began to swell and soon were swollen shut. Two weeks later vomiting began. The swelling of the lids disappeared and he returned to school after one month. In two days symptoms recurred, with swelling of the left eyelids and fever. He was relieved by the use of phenobarbital. Two weeks before examination the former symptoms recurred, with fever, headaches and considerable drowsiness.

The general examination gave negative information. Roentgenograms disclosed cloudy frontal sinuses only. Vision of the right eye was 6/15, and of the left eye 6/60. The pupils and reflexes were normal. The fundi showed bilateral choked disc of from three to four diopters, with pallor of both discs and absorbing exudate. The fields showed concentric contraction for form and color. The blind spots were enlarged. There was a cecocentral scotoma in the left eye. The neurologic examination was negative (see Figs. 2 and 3).

Exploration revealed an abscess of the left frontal lobe. Subsequently the vision improved to 6/10 in the right eye and to 6/15 in the left. The edema of the discs subsided to less than one diopter. The cecocentral scotoma receded to a small central scotoma in the

Date: Oct. 22, 1928. Record.



Fundi:

Fig. 3 (Case 2). Two years after patient's admission; form fields showed slight contraction for both eyes; partial restoration of color fields; the cecocentral scotoma in the left eye had receded to a small central scotoma; blind spots were normal.

left eye, and the blind spots were normal. Colors were recognized peripherally with both eyes.

This is the only case observed in the Mayo Clinic of abscess of the frontal lobe with bilateral choked disc and a central scotoma on the side of the lesion.

Phenomena of peripheral vision have the same significance in abscess of the brain as they have in tumor of the brain. The most characteristic alteration is hemianopia and is dependent on the site of the lesion. Homonymous hemianopia appears in temporal abscesses if they are subcortical and affect the optic radiations. It is

also found in abscess of the occipital lobe. Instead of complete hemianopia there may be only a quadrant hemianopia for colors, as occurred in Case 3.

Case 3: A girl, age 10 years, was brought to the clinic in May, 1928. She had had chronic otitis media in April. Since April 21 she had had headaches in the left temporal area. May 5, projectile vomiting with nausea developed. There was slight bulging of the right eye and diplopia. Choked disc of two diopters in the right eye and of three diopters in the left eye were found. The vision was 6/10 in each eye. Fields showed right homonymous hemianopia for colors.

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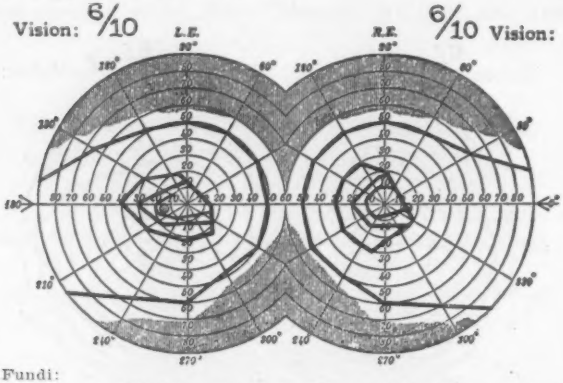


Fundi:

Fig. 4 (Case 3). Slight contraction of form field with incomplete right homonymous hemianopsia for color.

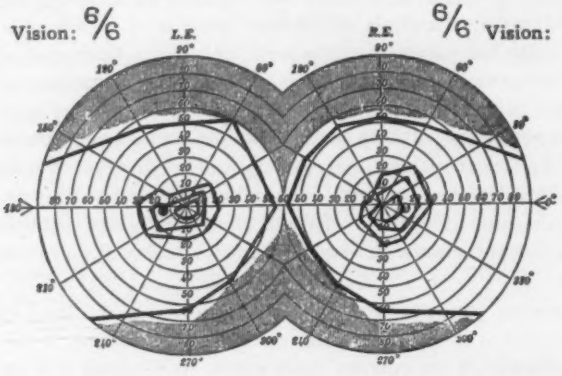
An abscess of the posterior part of the left temporal lobe was drained and the patient made a good recovery except for partial paralysis of the sixth and seventh nerves on the right side (see Fig. 4).

Characteristic field changes due to lesions of the frontal lobe are rare, and only occur in basally situated lesions. I have observed only one case (Case 2) of abscess of the frontal lobe with bilateral choked disc and a central scotoma on the side of the lesion.

Complete or incomplete homonymous hemianopia with choked disc is common in cases of abscess of the temporal and occipital lobes, whereas in abscess of the cerebellum and abscess of the occipital lobe complete bilateral amaurosis has been reported, probably the result of secondary optic atrophy. Uhtoff stated that hemianopia

accompanies 9 per cent of abscesses of the brain and that it occurs with greater frequency if the abscess is in the occipital lobe; such abscesses rarely originate in the ear. Hemianopia is sometimes the only symptom referable to the eye. Frequently, however, hemianopia is one of a number of symptoms among which choked disc is the most prominent.

Case 4: A man, age 42 years, had a history of having been injured while coupling cars. As a result of the injury, hemorrhagic suppurative cystitis developed. Subsequently he had a grand mal convulsion and on examination was found to have a few retinal hemorrhages in each eye. The vision was 6/6 in each eye; pupils,



Fundi:

Fig. 5 (Case 4). Bilateral left homonymous notching for colors in the upper quadrants; there was slight edema of the nerve heads; six days previously there was no edema of the nerve head and visual fields were normal.

pupillary reflexes and visual fields were normal. Six days later the vision was 6/6 in each eye, but there was slight horizontal nystagmus: both discs showed slight edema and there was left homonymous notching for colors in the upper quadrants.

A large mass, thought to be a tumor, was resected from the right temporal lobe. It proved to be a densely encapsulated abscess. The patient made a good recovery, although hemianopia persisted (see Fig. 5).

Hemianopia is of no diagnostic value in the differential diagnosis of abscess of the brain, but is of great value in determining the site and possibly the extent of the lesion. In case of bilateral suppurative mastoiditis with symptoms of abscess of the brain, the

visual field may be the only means of localizing the abscess. Nystagmus appears to be particularly common in cerebellar abscesses. It may be horizontal, rotatory or vertical.

Disturbances of motility which are the result of paralysis of one or more of the ocular muscles is seldom seen. Abducens paralysis has been found both on the same side and on the contralateral side of the lesion in cases of abscess of the frontal lobe and lesions in the frontoparietal region.

Signs of partial paralysis of the third nerve are most often associated with abscess of the temporal lobe and nearly always associated with choked disc. Ptosis, paralysis of one or more of the extraocular muscles supplied by the third nerve, paralysis of the pupil, or lack of accommodation have been reported as occurring singly or in combination. Paralysis of the third nerve is of considerable localizing value but paralysis of the sixth nerve may be concerned with only intracranial hypertension or inflammatory disturbances along the base of the brain, most frequently along the petrous portion of the temporal bone. It may lie far from the actual seat of the abscess.

Of the less frequently observed signs and symptoms, I have little to say because the observation is of little moment in the diagnosis or localization of abscess. Psychic blindness, visual hallucination, alexia, corneal anesthesia and changes in visual acuity are not common.

Conjugate paralysis with or without tilting of the head may be present, but is of little localizing value. The syndrome of the superior colliculus of the corpora quadrigemia, fixed pupils and paralysis of upward gaze indicate a lesion along the aqueduct associated with other signs of abscess, its occurrence is of considerable significance. It is frequent sequela of sleeping sickness.

SUMMARY.

The ocular manifestations that I have mentioned have been repeatedly noted in cases of abscess of the brain. Compared with data obtained at operation and at necropsy, their significance becomes apparent. Mos tconsultants have learned to recognize many indescribable changes in a patient in whom characteristic septic fever, drowsiness, headaches, vomiting, psychic phenomena and visual or other ocular changes develop in the course of a suppurative disease, all of which point in a general way to involvement of the brain. The neurologist usually must decide whether there has been actual invasion of the brain.

The ophthalmologist, then, has two very important functions. The first is to aid in localizing the lesion; his signs are choked disc, changes in the perimetric field, ocular paralysis and swelling of the lids and face. The significance of any of these observations varies with the dominant signs or symptoms in any given case. The second is to decide the proper time for surgical intervention. This is his most important service. An encapsulated abscess of the brain may be removed or drained with only slight risk, whereas if operation is performed before the abscess becomes encapsulated the patient seldom recovers. Here the degree and course of choked disc is stationary, the abscess, if present, is encapsulated, and the clinical picture becomes that of a tumor of the brain similarly situated. The ophthalmologist can then conscientiously urge surgical exploration.

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BRAIN ABSCESS FROM THE STANDPOINT OF THE OTOLARYNGOLOGIST.*

DR. WELLS P. EAGLETON, Newark.

Mr. President: I wish to thank you for the invitation to address this Society.

I have contended for a long time, in what teaching I do—for I am a clinician and not a teacher—that if I had anything to give, it was through some such method as the following: "Here is a patient with his history; we will now make an examination and from the history and examination, we will try to make a diagnosis and upon the diagnosis propose treatment."

But for myself, much that I have learned of real importance to me has come after I had made the diagnosis, and subjected the patient to treatment or operation, and the patient has died. I have then, at the postmortem table, reread the history, reviewed the examination, and in the presence of the actual pathology asked myself the questions:

"Was my diagnosis accurate enough for surgical purposes?" "Was there anything in the history or examination that should have suggested a correct or more accurate diagnosis?" "Was the surgery adapted to the control of the condition?" "Could my surgery possibly have done him any good?" And on the other hand, "Is there any evidence that the trauma of the operation or the technique adopted was the deciding factor in his death?"

And further and most important: "What can I learn from this case to help me in the diagnosis and care of future patients?"

With these questions in mind I have brought to this meeting 14 gross specimens of brains from patients who have died while in the Newark Eye and Ear Infirmary, or the Newark City Hospital; died as the result of suppurative intracranial complications of different kinds. With the specimens are the histories, which range from a complete history, such as we should compile, and do publish, down to the scattered memoranda of facts as they occur in our practice; the notes we make as we go along.

However, each specimen is accompanied by a complete report of all pathological findings observed during the postmortem. Each brain teaches a distinct surgical lesson.

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With the aid of these specimens, I shall take this occasion to add a few important things that I have learned about brain abscess since 1922, when I published "Brain Abscess."[1] At that time I believed everything that I therein stated; but, like everything else in life, I now know that the book contains many errors.

DEVELOPMENTAL RELATIONSHIP BETWEEN THE INTERMEDULLARY BLOOD SUPPLY AND THE MENINGES.

A biological fact that has profoundly influenced my views in regard to all suppurative lesions within the dura was the demonstration that, during the evolution of life at the same period when a blood supply entered the nervous tissues,[2,3] an intermedullary blood supply—there developed a covering for the brain and for the nervous tissue itself. This relationship between the intermedullary blood supply and the brain coverings exist from the rays and sharks (Elasmobranchii) up to man. This intimate relationship contains the secret of many of the surgical and diagnostic problems that are under discussion. For examination of the human brain demonstrates that every cerebral vessel is surrounded by a prolongation of the pia-arachnoid—a perivascular space. Also all the veins within the dura have no outer coat, they are simply endothelial cells; the place of the outer coat, the adventitial coat of the extradural veins is formed by this perivascular space prolongation.

Further, this anatomical relationship is of great regional importance; for an examination of the veins within the dura—the cerebral veins—where they pass from the brain into the sinuses or extradural veins, shows that there are several points in the neighborhood of the ear and nose which, were they to become the seat of suppuration in the bone would bring infection in contact with the coatless endothelial veins, directly through the veins themselves or by their associations[4] with the venous sinuses.

"POINTS OF VENOUS VULNERABILITY."

The minute veins that come out of the middle ear, and enter the sinus at the knee, and the veins of the vasa vasorum are especially important as routes of intradural infections. Two years ago I made a study of these veins and it considerably increased my mental horizon.

Venous channels and perivascular spaces are the chief routes of infection in brain abscess; for microscopical studies show that the intima of the cerebral veins and the perivascular space surrounding the vessels are the two most frequent routes of infection into the cerebral substance. For brain abscess may originate from either a retrograde thrombophlebitis or a perivascular space infiltration.

A chill clinically differentiates between a bloodstream invasion and a perivascular space invasion. Clinically the mode of invasion into the brain can be diagnosed and this may be of surgical importance, for when infection from the ear travels inside of the coat of the vein, there is always a chill; if it extends by the perivascular space there is no chill.

I show the microscopical section from the brain (see Fig. 1) of a man, with about the following history: He was operated upon in a general hospital for a right-sided mastoid suppuration. He then developed a lateral sinus thrombosis, which was also operated upon, following which he became slightly drowsy; he slept too much. He had a slight nystagmus on looking to the left but he had no papilloedema. His fields had been taken and they were said to be normal (see Chart 1). But as he was dopey, he was transferred to the Newark Eye and Ear Infirmary, and the day he was transferred our technician demonstrated indentations of the visual fields (see Chart 2). That same day he had a rise in temperature. Lumbar puncture showed the cerebral fluid to be infected. He died the day following the puncture. An autopsy revealed a hemorrhage, not only into the abscess, but into the lateral ventricle and the pons, which gave him his nystagmus and caused his death (see Fig. 1).

GROSS HEMORRHAGES IN BRAIN ABSCESS.

The inter-relationship between the perivascular spaces and the blood supply is still further shown by the frequency with which hemorrhages occur in suppurative diseases of the brain.

I show a specimen from a woman who had a cerebellar abscess. We explored and drained the abscess on two occasions. Suddenly, while apparently doing fairly well, she became comatose and died in a few minutes from a large hemorrhage into the arachnoid at the base (see Fig. 2).

I show a section from the brain of a woman on whom, in my effort to cure her of a profound sepsis from sinus thrombosis, associated with a meningitis, the result of a labyrinthitis, and a small brain abscess, I ligated the common carotid. Previously she had had intravenous injections of mercurochrome,[5] and as a result of the toxic action of the mercurochrome on the blood vessels and the disturbance of the circulation which the ligation occasioned, she had, on the following day, an interventricular hemorrhage (see Fig. 3).

And I have seen another woman on whom I operated and cured of brain abscess. Eighteen years later she went into coma and died; and as she became hemiplegic on the side previously paralyzed, I think that she had a hemorrhage into the old abscessed area.[6]

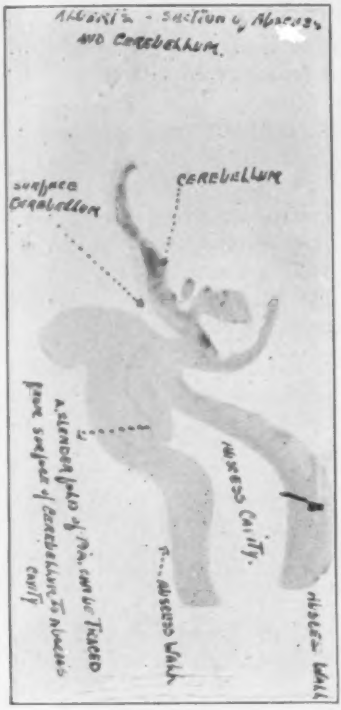

Section of Abscess and Cerebellum.
surface Cerebellum
CEREBELLUM
A SLENDER fold of Pia can be Traced from surface of Cerebellum to Abscess cavity
ABSCESS CAVITY.
ABSCESS WALL
ABSCESS WALL

Fig. 1. Case of hemorrhage in choroid plexus and into the lateral horn of ventricle, complicating temporosphenoidal lobe. The content report: "The dura is adherent to the posterior surface of the right temporal lobe. The adhering dura is continuous with the sinus when it makes the bend, and is directly over the area where the superior petrosal enters the lateral sinus.

"There is a large abscess, the size of a lemon, which has ruptured in the posterior end of the right temporal-sphenoidal lobe. The content of the abscess is hemorrhagic. There is a large vein running from the sinus to the abscess. There is a general encephalitis around the abscess. There is also a large hemorrhagic area in the posterior horn of the right lateral ventricle. (It was this hemorrhagic area that gave him nystagmus). The hemorrhagic area is one inch long and follows the large vessel of the choroid plexus, and nearly reaches the pons in the location of the basilar artery. Undoubtedly the perforation into the lateral ventricle caused death." Microscopical examination shows a retrograde thrombophlebitis through a small vein from the abscess. The meninges are densely infiltrated by leukocytes, mostly polynuclear, and contain large thrombosed vessels. A slender fold of pia can be traced from the surface of the cerebellum to the abscess cavity. The fold contains thrombosed blood vessels.

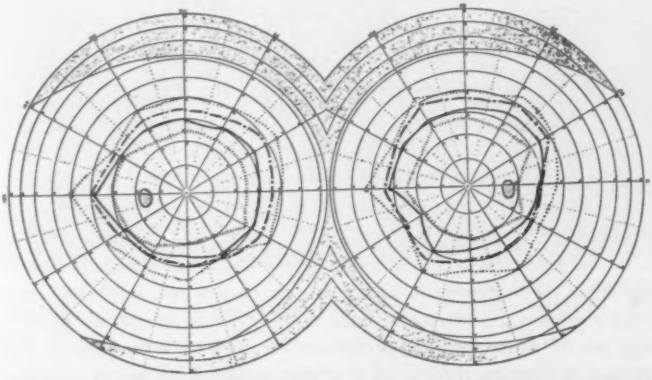


Chart 1. Showing failure, by imperfectly trained technician, to elicit a proper field in a drowsy patient.

SPECIFIC SURGICAL LESIONS IN INTRADURAL SUPPURATION.

What are some of the other lesions of vascular origin found in these 14 specimens? First of all, the frequency of infarcts.

a. Infarcts: A child has earaches, it vomits and is ill. The doctor does a double myringotomy: one ear discharges freely; the other, little or not at all. But the child continues sick; has temperature and vomits again, and on the fifth day after the original earache, he suddenly goes into coma, becomes hemiplegic, has a lateral deviation of the eyes away from the side of the suppurating ear and has repeated convulsions. At autopsy we find a hemorrhagic infective infarct, undoubtedly from the ear, that had occluded one of the large cortical veins, around which occlusion there is an area of hem-

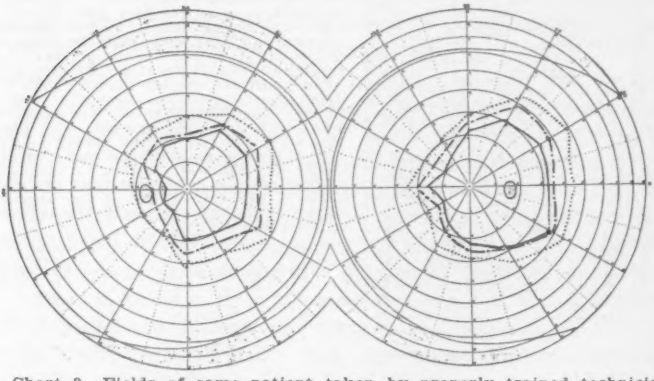


Chart 2. Fields of same patient taken by properly trained technician after repeated questioning. Patient still dopey.

orrhage—while the pia-arachnoid mesh of the cortex is covered by hemorrhagic areas (see Fig. 4).

Such infarcts are relatively common. They may get well—many do—if the vascular occluding nidus is not infected or when the infection is overcome by the protective reaction of the surrounding arachnoid.

To the latter class belong some of the so-called "pseudoabscesses," in which the surgeon, in the presence of definite localizing symptoms, explores the brain and finds nothing. The operation, however, may be of some benefit, providing the operative trauma is slight because it liberates the excess of cerebrospinal fluid which is secreted as the result of the "protective" reaction.

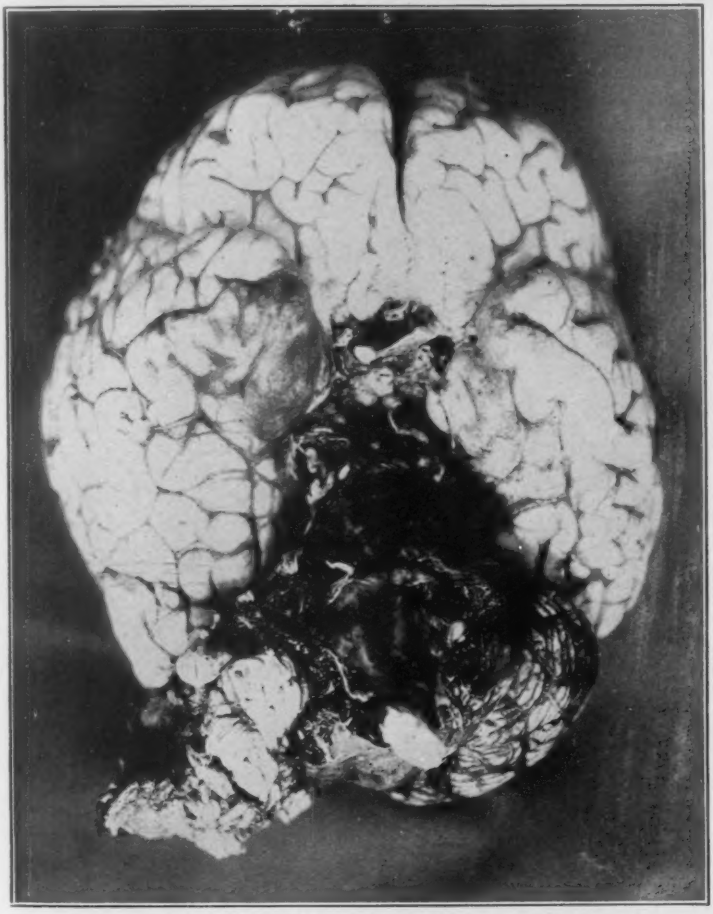


Fig. 2. Large subarachnoid hemorrhage filling basal cisterna and causing death; an unusual vascular complication of cerebellar abscess.

But if the surgeon is one of the brilliant, hurry-up kind that poke holes in all directions through the dura in the brain in an effort to find a pocket of pus within the brain—the patient dies.

But septic infarcts do not always get well; nor do they immediately cause a severe meningitis, as in the case of the boy.

An infarct in the substance of the brain may remain quiet for some time and then develop a brain abscess.

I show a section of the brain (see Fig. 5) of an elderly woman, who gave this history: She had had a left-sided chronic running

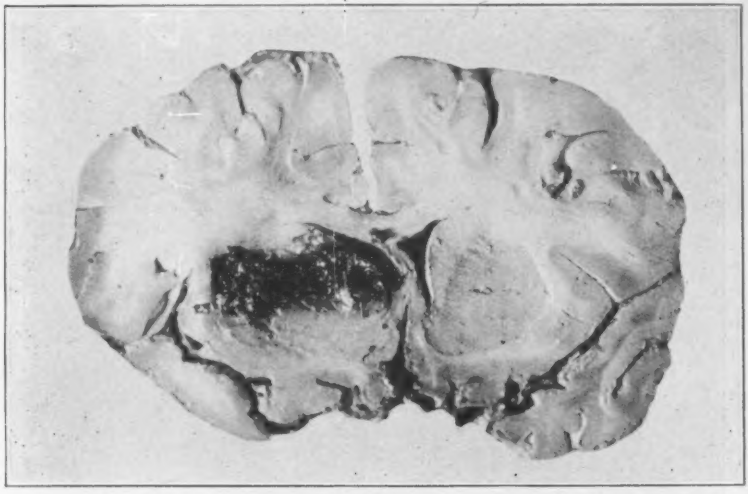


Fig. 3. Large intraventricular hemorrhage from a small abscess and ulceration of a branch of the middle cerebral. The intraventricular hemorrhage occurred 24 hours after the ligation of the homolateral common carotid; the vessel walls have probably been damaged by the intravenous injection of mercurochrome. The mercurochrome injection was followed by a chill, frequent urination and mercurochrome in the urine.

ear for 30 years and recently had a convulsion. But her daughter said she had had convulsions a number of years before. A neurological examination revealed nothing.

Because she had repeated attacks of pain in the left ear, in which the labyrinth had long ceased to function, we did a radical mastoid exenteration. On the fifth day after the operation, she had another convulsion, and on becoming conscious she showed a right hemiplegia and an aphasia. But the following day she was

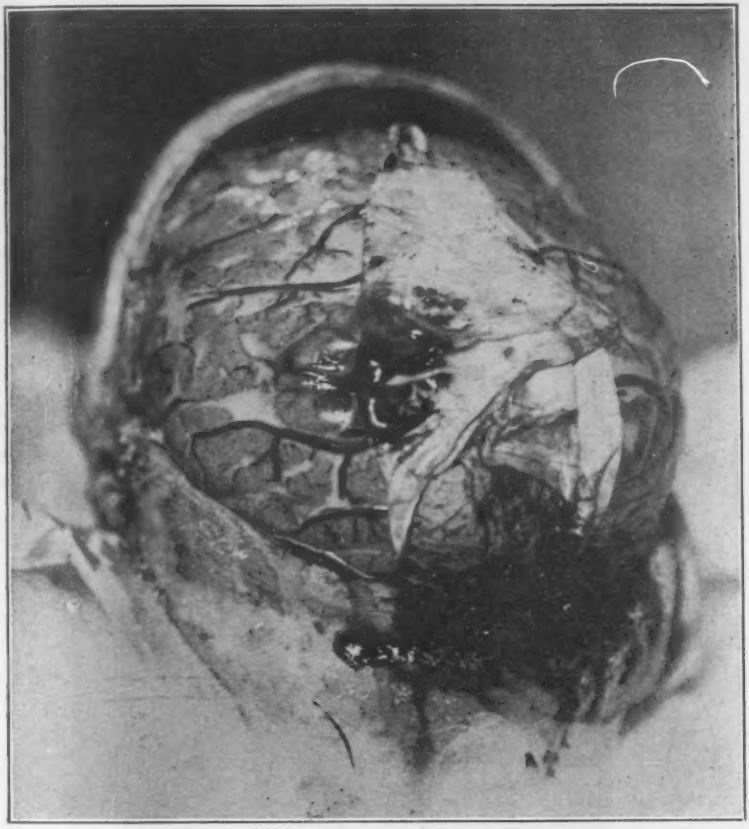


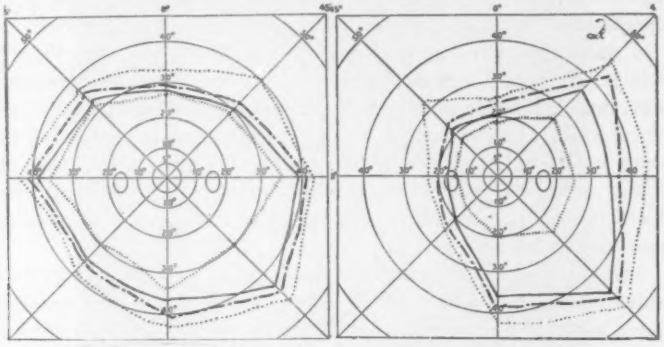
Fig. 4. Hemorrhagic infarct in the midst of a general suppurative leptomeningitis. The infarct is in reality a terminal manifestation of a virulent pneumococcic (Type III) meningitis. The meningitis had produced no alarming symptoms until the vascular occlusion and associated hemorrhage occurred.

well; apparently all symptoms had disappeared. She went home—about three weeks afterwards—seemingly in good health. While at home her daughter said that she had some headache but it was not severe enough to cause her to return to see us. Suddenly she became hemiplegic and went into coma; she was brought back to the hospital—an outstanding case of brain abscess. The abscess was evacuated but she died of hemorrhage into the surrounding tissue.

Fig. 5. Abscess in neighborhood of internal capsule, the result of small septic infarct from a suppurating ear. The abscess did not develop for many weeks after the deposit of the infarct.

Infarcts, although far from uncommon, are but infrequently recognized. They may give all kinds of transient aploplectiform symptoms, depending on the area disturbed by the vascular occlusion.

I saw a patient, in consultation with Dr. Walter Tymeson, who, several days after a simple mastoid operation, followed by an up and down temperature, suddenly had repeated left-sided convulsions. On regaining consciousness there was demonstrable by the rough finger test a bilateral left hemianopsia which was complete up to the fixation point. However, on the following day the hemianopsia had disappeared from the field of the left eye, leaving only an indentation in the field of the right eye—the side of the lesion—this also soon disappeared and never recurred (see Chart 3).



Left Eye. Right Eye.

Chart 3. Left-sided and upper indentation of visual field of right eye; the only remains of a complete bilateral left-sided hemianopsia of the day before, in a patient suffering from a vascular occlusion (left-sided convulsions), a complication of acute sepsis from mastoiditis and suppurative otitis.

When, however, an infected infarct is deposited into the brain's substance it may be quiet and then later develop an abscess, as in the case of the elderly woman (see Fig. 5).

b. Subdural Space Abscesses: The second surgically interesting pathological process revealed by these 14 specimens is the frequency of abscesses localized in the subdural space. They have not been emphasized as they should be; for they are of frequent occurrence, especially over the frontal lobe. In my experience possibly 25 per cent of all the frontal lobe abscesses are subdural space abscesses and if properly sought for, they should be easily located. A large osseous opening and a dural flap over the proper area will disclose a flat collection of pus on the surface of the brain. It is apt to

extend here, there and apparently everywhere. On the other hand, the passage of a brain searcher through a small dural opening may fail to reveal pus. I have more than once, and that in recent years, overlooked a thin subdural collection—a so-called "superficial abscess," because I had explored through too small an opening in the bone and a nick in the dura.

In the temporosphenoidal lobe subdural space abscesses are not common; however, they are fairly frequent over the tentorium under the occipital lobe.

All cases of infection through the ductus endolymphaticus—so-called "empyema" of the ductus endolymphaticus"—are in reality intradural space abscesses which are apt to become subdural space collections. Early recognized and properly operated on, ductus endolymphaticus cases should give a high proportion of recoveries.

I show a specimen from a case of sinus thrombosis[7] (see Fig. 6), which had been operated on and was apparently cured, but later developed cerebellar symptoms. We opened an abscess in the subdural space of the posterior fossa, and accidentally perforated the dura of the middle fossa; pus escaped. The sinus infection, by direct continuity or a retrograde thrombophlebitis of a small vein, had originated a subdural space suppuration in both the posterior and middle fossae.

Cases of erosion of the superior semicircular canal are apt to cause subdural abscess; and cases in which during a mastoid operation there has been an injury to the dura may develop subdural abscess. In the latter connection my experience shows that, if after drainage of a temporosphenoidal lobe abscess the case does not do well, the possibility of a collection of pus in the subdural space incident to the operative opening of the subdural space is to be considered.

All cases of subdural space suppuration require large dural incisions as the infection may spread freely by continuity because of lack of adhesions between the inner surface of the dura and the outer layer of the arachnoid.

Aids to Diagnosis in Brain Abscess: What have I learned about diagnosis?

I place more importance than ever, in diagnosis, upon the probability that certain areas in the primary focus of infection if diseased —that is, caries in Trautman's triangle; labyrinthitis; perisinus abscess; granulations *over* the sinus low down towards the bulb; jugular bulb thrombosis—are apt to follow definite paths when the suppuration extends. That is the importance: 1. of *anatomically known routes through the dura* by pathological and anatomical preformed ways in the arachnoid and into the brain:

Consequently, the importance of: *2.* the *site of the original infection*[8] as found during a simple or radical mastoid operation performed at a time when there are no brain symptoms; because if brain symptoms occur later, the pathological change revealed by the primary operation may suggest what to do and where to go.

A Nonfunctioning Labyrinth in Diagnosis: The labyrinth stands in the middle of the petrous pyramid, so all osseous paths of infection are either *a.* extra labyrinthine, *b.* intra labyrinthine, or *c.* in

Fig. 6. Subdural space suppuration in the posterior fossa with an associated middle fossa subdural space collection of pus, both originating from a retrograde thrombophlebitis of veins from an infected lateral sinus. Mistaken for an intracerebellar abscess.

the petrous apex. Consequently, of prime importance in the localization of the site of an intracranial lesion is to determine, "Is the labyrinth functioning?" and, if not, "when did it lose its function?" For if the labyrinth has recently ceased to function, the path of infection is probably through the labyrinth and has entered the dura by the internal auditory meatus.

3. The Importance of Vomiting? I know that some of my friends think I am extreme in this, but I believe that, given a suppurating

ear, if the patient vomits, something has happened within the dura. Of course, it is possible to have an abdominal cause, but ordinarily, especially in children, unless there is a manifest reason, vomiting during an otitis media should arouse the suspicion of an intracranial complication.

Again, our experience teaches that in a considerable number of cases the times of invasion dates from an initial vomiting. The case I showed (see Fig. 4, the child with pneumococci infarct) had an initial vomiting which was one of the manifestations of a localized meningitis and which was probably curable at this time by the complete exenteration of the primary focus in the mastoid, or even during the next day or so if the mastoid surgery had been associated with evacuation of an adjacent collection of infected cerebrospinal fluid. Five days later, however, when there were septic infarcts and general suppurative leptomeningitis, the case was hopeless.

4. Routine Visual Fields: We have a visual field of each eye taken every day. It is a tiresome procedure but I have learned that the chief manifestation of the *edema which accompanies a brain abscess may be alterations in the visual fields.*[9] However, like all edemas, the indentations may vary from day to day; they may be present one day but gone the next day. The only time when fields can be properly taken is when there is only a possibility that an abscess may be present; when the intracranial lesion is manifest the patient is usually so drowsy that he cannot co-operate.

I exhibit 78 fields taken on one case of brain abscess that recovered.[10] The case is remarkable, in that this boy's brain was explored and drainage instituted on four occasions; each time largely because the sudden alterations in the fields suggested that the suppurative process was again advancing. The patient himself came to appreciate when he was not doing well by the condition of his fields, even in the absence of other symptoms; he would say, "Doctor, my fields are in today. I suppose you will be operating on me again."

But homonymous indentations of the visual fields may not be present and still there may be extended edema of the brain, provided there is no intradural pressure. This may occur when a decompression operation has been performed. For it is the pressure upon the visual tract by the edema of cerebral tissue confined within the dura and skull that causes the indentations.

I show the brain of a man (see Fig. 7), who had a left frontal sinus suppuration, then a cavernous sinus thrombophlebitis and a meningitis. I ligated the common carotid, eviscerated the orbit and drained the septic cavernous sinus to control his bloodstream infec-

tion; I then removed the bone over the whole left frontal area and made a small opening in the dura; thus giving him a large bony decompression and evacuating a collection of septic fluid to combat the localized meningitis. For a long time he did well. But he had a slight aphasia at times. He also was depressed; but he had no headache. We took many visual fields, and largely because they all failed to show any indentations he was allowed to leave the hospital after

Fig. 7. Large abscess in left frontal, parietal and temporal lobes, but causing no hemianopic indentation of fields because of extensive decompression of left frontal region.

several months (see Fig. 8). Three months later he returned with headache, vomiting and papilloedema. We explored and found an arachnoid cyst; and we again thought we had helped him. However, one day he went into deep coma, and he died of a very large abscess which had failed to cause indentations of the visual fields because of the extensive decompression.

5. *The Value of Nystagmus:* I think those of us who do this work should throw out of our minds much that has been taught about the localizing value of the direction of the nystagmus. I am speaking now of intracranial lesions, not labyrinth disease. We

should ask ourselves: Has this man a spontaneous nystagmus, or has he not? We know too little about the central connections of the vestibular apparatus to befog the issue by clinging to theories that we now know are wrong.[11] I am sure that we frequently confuse and deceive ourselves in trying to make a diagnosis of the *location of the lesion by the direction of the nystagmus.*

Fig. 8. Large frontal bone defect, lower one-half of which was granulating mass several months after operation, following evisceration of orbit, draining of cavernous sinus, and evacuation of septic fluid from meninges. As the result of the extensive decompression there were no indentations of the visual fields, although a large abscess was present (see Fig. 7).

There are a few points, however, that have stood me in good stead about nystagmus in suppurative lesions.

a. Vertical nystagmus, either spontaneous or induced, suggests a brain stem involvement.

b. A changing spontaneous nystagmus suggests a retrolabyrinthine origin.

c. A vertical diplopia associated with or *followed by a spontaneous nystagmus*[12] suggests a very deep, a midbrain collection in the anterior midbrain segment of the posterior fossa—mesencephalic.

Of abnormally induced nystagmus, the most important in acute suppurative lesions is that during a cold caloric is *d.* great diminution or abscence of nystagmus in the upright position, especially of the contralateral side, associated with a normal nystagmus in the horizontal position[13] is diagnostic of increased intracranial pressure.

But they are all only suggestions; they are not really localizing, for the nystagmus may originate from a lesion above the tentorium.

I exhibit the brain of a man who was referred to me by Dr. Moorehead, of Brooklyn. This doctor had done a very clever thing. He said this man has brain abscess, and when I asked why, he said: "Because his tongue has remained coated too long, and now he has a little nystagmus and has a pain in the head." I thought "that is easy—he must have a cerebellar abscess;" but when I examined the patient, it was not so easy. He had had a double mastoid—the right lateral sinus had become infected and had been operated upon. But he now had pain in the left occipital region and a changing nystagmus. We decided that he had a cerebellar abscess, probably on the right side, although his pain was over the left occiput and in the left eye, which made a left-sided lesion a possibility. We did a double cerebellar decompression and explored the cerebellum and found nothing. The first day after the operation, the nystagmus disappeared. But the man did not improve. His fields were not indented; but suddenly (when he was almost unconscious and somewhat irrational) the technician said she thought he *could not see on his right side.* That suggested that the abscess was not in the posterior fossa but in the middle fossa and *on the left side.* He died, and the specimen shows the abscess in the occipital lobe of the right side.

MISTAKES.

Now, what have we learned about what we should not do?

Danger of Lumbar Puncture and Ether in Brain Abscess: I do not do a lumbar pncture in any case in which there is even a suspicion of an abscess which may involve either the posterior fossa or any part of the midbrain cone that passes up through the tentorial ring. I exhibit the brain of a man who I thought had a meningitis and on whom we performed several lumbar punctures and two ventricular taps. Suddenly after a lumbar puncture he stopped breath-

ing. At postmortem we found a small hemorrhage into the arachnoid over the medulla that we had caused by the last lumbar puncture.

Last of all, we do not give our cases ether. We try to do all operative work under local anesthesia. I show the brain of a man sent to me by Dr. Lynn Emerson, of Orange. His history was that he had had two running ears for some months, then they ceased to discharge and now he had pain in his head, vomiting, nystagmus

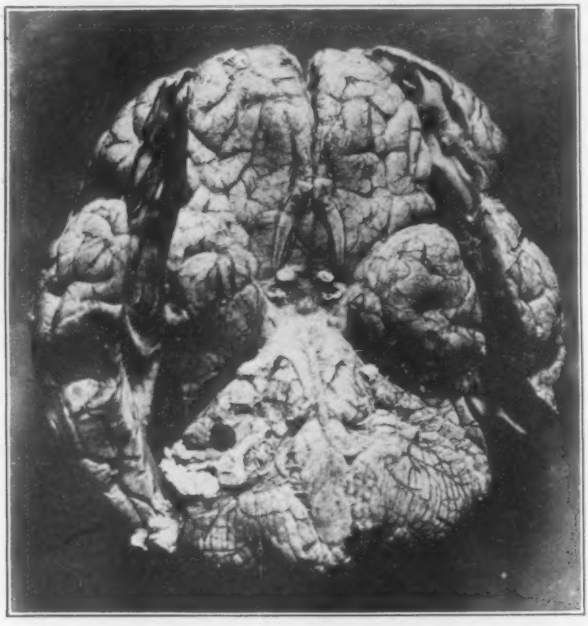


Fig. 9. Area of encapsulated pus in the meshes of the arachnoid marked by black disc.

and papilloedema. His fields were not indented. We tapped both ventricles, and while I was "playing with him" he became so irrational that he could not be controlled and he was given ether, preparatory to a cerebellar exploration. Suddenly he stopped breathing, and although artificial respiration was performed for five hours and although with a direct laryngoscope his cords were seen to move with each artificial filling of the chest, he never breathed again. At autopsy we found a small abscess (see Fig. 10) in the arachnoid

mesh near the angle; and later another small abscess near the midline, which was so hidden that we did not find it until we had hardened the brain—a little abscess in the substance of the brain stem, that had given tremendous symptoms and caused death by respiratory paralysis when ether was administered (see Fig. 9).

Operative Results: What has been our mortality during the last seven years in the treatment of brain abscess? I must confess that in the temporosphenoidal abscess our results have not been as brilliant as those reported in the literature, nor have they greatly improved over those of former years. This I think may be partially due to may having abandoned the large bony flap that I originally employed.

But in the very nature of the pathological process and its anatomical situation—an abscess within the temporal lobe must be a "killing" disease in a large proportion of cases. For it is a destructive suppurative space-consuming lesion, confined within a bony cavity; and situated not only in the highest organized tissue of the body —the brain—but in the latest addition to the neopallium—the sphenotemporal lobe—in the neighborhood of the basal ganglia and the central ventricular system, none of which can be displaced without serious damage to the ganglia of extension of the suppuration into the ventricle. Consequently, although a temporosphenoidal lobe abscess is in a symptomatic "silent area," the above unfavorable factors make such an abscess especially liable to a surrounding encephalitis and infective thrombophlebitis; while its proximity to the ear renders a relighting of infection in the damaged cerebral tissue a constant possibility.

I wish we followed all our cases of so-called cures of abscess of the brain for three years at least; the same as they do in cancer cases. I think we would be surprised to find that several had died with cerebral symptoms. Such has happened to two of my cases[14]; in which, after over a year of apparent health, operation or post-mortem[14] revealed an area of suppuration within the brain which had been quiescent until reinfected.

But our results in cerebellar abscess have markedly improved—over 50 per cent have recovered. This is largely due, we think, to the extent of the initial bony decompression; for the first thing we do is to remove all the bone covering both cerebellar hemispheres from the opposite lateral sinus to the diseased petrous pyramid and from the transverse sinus above to the foramen magnum below[15]; and as this is followed by "unlocking" of the petrous pyramid[16] with

removal of the angle of the superior petrosal and of a large part of the squama, the bony decompression is very large.

Also intradural suppuration of the frontal region has given much better results than before. Histologically this should be so, for here we have a localized suppuration in a "silent" area from the ventricu-

Fig 10. Same brain as Fig. 9. Encapsulated abscess, possibly tuberculous in brainstem so small that it was not discovered at time of post-mortem—respiratory failure on administration of ether.

lar system and basal ganglia, without firm connections to the dura and thus amenable to considerable displacement without damage to the surrounding parts. In addition to this the proper short-circuiting of the mucous membrane[17] of the nose should render a later infection much less liable than in the temporal lobe.

Lastly, our results in meningitis have been such that we have been more than encouraged. In the last 107 cases that Dr. John V. Bissett and myself have operated upon, 62 have recovered—about 60 per cent.

Today if we have a case of abscess of the brain which develops meningitis, we pay little attention to the abscess, but try to cure the meningitis. For, as long ago we learned that when a patient with a chronic running ear develops intracranial symptoms, not to perform a radical operation, but to attack the intracranial lesion; so recently we have realized that in meningitis resulting from an abscess the meningitis should be first controlled, and this, if successful, will allow of the drainage of the abscess with less risk to the patient.

Conclusions: In conclusion, I want to emphasize: *a.* Every otologist should, for his own sake, make a careful note of each pathological finding in all cases of mastoid; and later if intracranial symptoms develop try to think anatomically. With infection in such and such a place, he should try to visualize what has happened inside the cranium; what course the process should take in its passage through the dura, and within the arachnoid or cerebral tissue. *b.* Vomiting in any case of a running ear should make the surgeon suspicious of an intracranial complication. *c.* Every chill that occurs in an intracranial complication is a sign of bloodstream infection, although the infection may be in one of the veins of the cerebral tissue, not necessarily in a venous sinus. *d.* Have somebody take visual fields daily. It is a difficult task; do not try to do it yourself or it will not be done. Do not ask an ophthalmologist to do it, for he will do it but once. But have someone whose calling it is to take fields routinely, carefully and intelligently; because routine fields are just as important, in my opinion, in the early diagnosis of localized intracranial suppuration as a temperature record. *e.* Do not fuss about the direction of the nystagmus. Observe each day whether the patient had any spontaneous nystagmus or not. Decide that it is retrolabyrinthine, and use that as a basis. *f.* If he has no spontaneous nystagmus, ascertain if the vestibular manifestation of increased intracranial pressure is present. *g.* Do not do a lumbar puncture unless you are sure the abscess does not involve the posterior fossa or the midbrain cone. *h.* And do not administer ether when unnecessary if you think you have brain abscess.

If we are to advance we must realize that nobody knows much about this work. To do it well, we should organize teams of two or three men, preferably young men, with good surgical training,

who have time and inclination to do a work which is nerve-wracking and laborious, but most intensely interesting. Such a team in the different eye and ear hospitals would soon develop a diagnostic and operative technique, would rapidly elucidate the factors and problems of intradural suppuration, and save many lives which are now sacrificed because of our imperfect knowledge.

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15 Lombardy Street.

BRAIN ABSCESS FROM THE STANDPOINT OF THE BRAIN SURGEON.*

Dr. William J. Mixter, Boston.

The viewpoint of the neurological surgeon in regard to brain abscess must of necessity be somewhat different from that of the ophthalmologist or the otolaryngologist. Fortunately this difference is more superficial than real, for the co-operation between these branches of surgery has been close in the past. It must become even closer in the future if we are to continue to advance in our treatment of this serious disease. To this end we must not only compare our results, we must reconcile our differences and also see and study each other's ca es. Finally, we must each of us adopt the advances made by the other, scrapping our own methods wherever we can find better ones.

Let us first consider the allied subjects of pathology and etiology. Brain abscess for the purposes of this discussion may be defined as any localized collection of pus lying within the cranial cavity. (This definition will exclude diffuse inflammatory processes such as encephalitis and meningitis, except in so far as they are associated with true abscess, and will also exclude thrombi except as they are associated with abscess. The pathologic process thus is an abscess lying within the cranial cavity, caused by any one of innumerable microorganisms or by a mixed infection.) The infectious organisms most commonly found would include the streptococcus, staphylococcus, pneumococcus and a mixture of these with other organisms, particularly the colon bacillus Few surgeons realize how frequently the colon bacillus is found in brain abscess, nor how frequently the pus from a brain abscess may be sterile. These, I believe, represent our usual findings, especially if the abscess is secondary to an infection arising in the realm of the otolaryngologist, but we must be eternally on the watch for multiple or dumbbell abscesses or for strange infections with bizarre clinical pictures and unusual operative findings. I believe that you gentlemen whose work is primarily devoted to nose and throat diseases see as many or more abscesses than we whose work is primarily neurosurgery, but that we see more abscesses arising from structures other than the air passages. Among these other infections may be mentioned tuberculous abscess, abscess

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due to yeasts, and finally those due to fungi, such as streptothrix and actinomyces. These fungus infections have forced themselves to my attention during the last few years. A recent experience was of particular interest:

The patient, an elderly man, had had severe right facial pain for several weeks following extraction of some teeth under local anesthesia. The pain came on very promptly following the first attempt at extraction, which was unsuccessful owing to lack of anesthesia following novocain injection. After some weeks the patient developed an orbital abscess, which was opened and drained but did not subside. The process extended gradually with brawny swelling about the eye and exophthalmos, but without elevation of temperature. The next development and the one that brought him under my care was the appearance of signs of increased intracranial tension and right-sided cortical irritation. Operation revealed a small abscess beneath the anterior portion of the temporal muscle, marked thickening and infection of the underlying bone and of the meninges, and an area of porky hardness in the temporal and frontal lobes, in which were several small abscesses. Examination of the pus showed actinomycosis. Autopsy revealed that the infection involved the base of the skull, apparently entering at the foramen ovale or rotundum. The infection had apparently traveled up the nerve sheaths from the jaw to involve the base of the skull.

Such experiences as the above bring us all to the realization that brain abscess is not a simple lesion, but one in which the causative agent may be varied. Other cases might be quoted as follows:

A child with an influenzal abscess and meningitis following a simple depressed skull fracture; an old gentleman operated on for brain tumor with no thought of abscess, who in reality had a metastatic brain abscess, and a case operated on by my associate, Dr. Hodgson, that showed a tuberculous abscess of the frontal lobe without other demonstrable lesion. Such cases as these are less often seen by the otolaryngologist and seldom by the ophthalmologist, except the case of actinomycosis.

We neurosurgeons must remember, nevertheless, that brain abscess is usually caused by inflammatory disease in the air passages, and that we must look to you to help us wherever this occurs. We must *all* remember that suppuration anywhere in the body may cause brain abscess, though of course suppuration in the lung is probably the most common primary focus after the upper respiratory tract. Bloodstream infection is another common cause, but even in the absence of any evidence of septicemia we may have metastatic brain abscess from such distant foci as pelvic abscess and the like.

We must remember that the character of intracranial infection due to adjacent inflammatory disease will vary considerably according to its location. Infection of the frontal sinuses is the commonest cause of osteomyelitis of the frontal bone. This disease is frequently fatal and its course rapid. Should it become chronic or even subacute, frontal lobe abscess is a common complication. Infections of the ethmoid and sphenoid cells, on the contrary, seldom result in abscess. Diffuse meningitis is the rule, and if abscess does occur it is almost invariably a subdural or subarachnoid abscess rather than infection within the brain itelf. If we continue from before backward we come to the most common brain abscess, that of the temporal or parietal lobe. Such abscesses arise usually as the result of middle ear disease and it is in these cases that we may find the process occurring by direct extension. I think, however, that abscess from direct extension is far less common than was taught a few years ago and that infection from septic thrombi is more usual. Finally, we come to cerebellar abscess. Acute cerebellar abscess presupposes a coexistent thrombosis as a result of mastoid disease and does not occur by direct extension. Chronic cerebellar abscess presupposes a pre-existing sinus infection which may be healed. These facts must be borne in mind in the treatment of these various types of abscess.

When we come to the question of diagnosis of brain abscess we find that we must not depend on our own efforts alone but must call for the co-operation and knowledge of a varied group of consultants, of whom the neurologist is the most important.

In many instances diagnosis is clear with typical history of focus of infection, followed by increasing signs of intracranial extension, including headache, somnolence, etc. Examination reveals choked disc, changes in the visual fields, cerebrospinal fluid changes and evidence of weakness of one side of the body, or cortical irritation. With such a case we have no difficulty, but there are many atypical and puzzling cases that do not present the classical picture of abscess. Take for example the differential diagnosis between brain abscess and brain tumor. These two diseases may present at first glance an almost identical clinical picture. Where, then, may we find important differences? Brain tumor is a noninflammatory disease, and abscess is inflammatory; yet the temperature chart may be flat normal in both cases. The white count, however, is often a clue. A persistent leukocytosis, particularly with an increase in the polymorphonuclear leukocytes, is very suggestive of abscess, and on this alone the diagnosis sometimes hinges.

The cerebrospinal fluid picture is more complicated but is of no less importance in arriving at a differential diagnosis. Pressure relations, chemistry and the cellular elements are all important, and for this reason we must insist on a complete and thorough examination of the fluid if lumbar puncture is not contraindicated by evidence of marked increase in intracranial tension and compression of the medulla.

Meningitis is of course easily differentiated by the presence of polymorphonuclear leukocytes and by the decrease in chlorids; hence the diagnosis of abscess with complicating meningitis, as against tumor, is readily made. Even the presence of a small number of polys is usually a very strong indication of abscess. In both tumor and abscess the pressure is increased, protein is increased, and if there is no meningitis, chlorids are normal. An increase of lymphocytes is strongly suggestive of abscess as against tumor, but not absolutely positive, as we have recently discovered that there are a few tumor cases that show a similar increase in lymphocytes. Thus we must not make a diagnosis of abscess as against brain tumor solely on the presence of lymphocytes. We can say such a diagnosis is probable, but in order to make it certain we must have other presumptive evidence of abscess either in history or the physical examination.

Syphilis and tuberculosis will give a similar cell picture, but these diseases can almost always be differentiated by other means.

Dr. Eagleton has stressed the importance of fundus and visual field examination in abscess. We should take the lesson to heart for here is a diagnostic measure which not only gives us important means of localization, but also gives us perhaps the optimum time for operation. The differential diagnosis between sinus thrombosis and abscess has been freely discussed of late and is far more in your province than in mine. From the viewpoint of the neurological surgeon, Tobey and Ayers' test for sinus thrombosis by compression separately of each jugular vein would seem to be logical and accurate. In the few cases in which I have seen this test used it has been of definite value, and I know from experience that in tumor this test is not positive. Some confusion may be caused in the rare case where jugular compression causes temporary cessation of the heart beat from stimulation of the vagus. This occurrence is rare and is quickly recognized by the fact that the radical pulse stops. The heart starts beating again as soon as compression is released.

Differentiation between a slowly leaking abscess, particularly a superficial abscess, and meningitis is at times difficult and often

impossible. Even an increase in intracranial signs with improvement of the cerebrospinal fluid findings may not tell the story for a certain amount of walling off at the level of the tentorium or below may give the same picture in diffuse meningitis as in abscess. Of course here we are faced with the question—when does walling off of a diffuse meningitis constitute an abscess?

The treatment of brain abscess is essentially surgical but there is nevertheless a good deal of difference of opinion as to how and when. To my mind one of the very important considerations is the original focus, especially if such focus be demonstrated in the nasal sinuses or mastoid. This should be cleaned up as rapidly and completely as possible. Frequently it is wise to do this before attacking the intracranial lesion, particularly if the position of the latter is uncertain or if it is of recent and rapid development.

I am not one of those who believe that a probable case of brain abscess must be explored at once and the abscess drained at all hazards. I believe that as soon as an abscess can be satisfactorily localized a careful but determined effort should be made to evacuate it, but I am firmly of the opinion that many patients have been lost as the result of ill-planned and ill-executed search for a brain abscess before such an abscess had been given time to indicate its position. Better a certain amount of delay rather than a brain lacerated and infected by too enthusiastic exploration.

During the investigation of the original focus, the surgeon should watch closely for a lead. If one is found he should follow it carefully through skull, meninges and brain and drain the abscess wherever possible through the original focus. For this reason I am firmly of the opinion that abscess of this nature should be handled by an otolaryngologist. There is, however, one very important proviso, namely, that the otolaryngologist be conversant with modern methods in neurosurgery and by training and practice able and willing to apply them. If such an operation is performed by one ignorant of these procedures, the result will probably be deplorable. It is better to wait for a trained consultant than to plunge in ignorance. We at the Massachusetts General Hospital are very fortunate in our close affiliation with the eye and ear infirmary and the close proximity of the two institutions. We are often able to combine our forces to good advantage, and have occasionally been able to carry out the following plan: The exploration of the original focus and, if possible, drainage of the abscess is carried out by the otolaryngologist. Then if a free exploration of the brain through a clean field is indicated, the neurosurgeon, who has been present from the beginning,

can step in with a complete fresh layout and carry on without loss of time. In many institutions such co-operation is not possible. Under these circumstances the surgeon, be he otologist or neurosurgeon, must be prepared to carry out both procedures. On account of the special training and technique necessary for such operations as exploration of ethmoids, sphenoids or mastoid, the otolaryngologist should usually have the preference if the operation is to be carried out entirely by one man.

We have already spoken of the etiology of abscess, particularly the fact that some of them are caused by inflammatory processes in the nasal sinuses or mastoids, and others are frankly metastatic. We have also spoken of the advantages of exploration through the original focus if this is possible. Here is a word of warning. The neurosurgeon must take care not to overlook the possibilities of direct extension and the otolaryngologist must investigate his cases from all angles, that he may not overlook some distant primary focus.

If a brain abscess has been located through the original focus we must assure ourselves that drainage is adequate before we stop. In many parts of the body partial drainage of an abscess cavity may effect a cure, but this is not true in the brain. There are many factors that contribute to this, and among them may be mentioned the formation of a dense wall of inflammatory tissue about the chronic abscess, the lack of power of resistance and repair in brain tissue, and the well known tendency of brain abscess to burrow deeper toward the ventricle, rather than toward the surface. Thus it is frequently necessary to drain the abscess *de novo* through a clean area even if it has occurred by direct extension. Drainage through a clean area is almost always necessary in subcortical metastatic abscesses.

In this matter of exploration and drainage through a clean area we find that both the otolaryngologists and the neurosurgeons have contributed to our knowledge. To men like Cushing and Frazier we owe the technique of modern brain surgery, but the more recent advances in exploration and in the technique of drainage we owe to Eagleton, Mosher, King and others. Cerebral abscess can be searched for in one of two ways, either through a small drill hole or a good-sized bone flap. The choice of type of operation depends somewhat on the definiteness of the localizing signs, condition of the patient and location of the abscess. I personally have a leaning toward exploration through a drill hole, particularly if the patient is in poor condition. A small nick only is made in the dura and the exploration is made with a large, round-pointed Cushing needle.

Great care should be taken not to puncture the ventricle. (Punctures are made in various directions and to various depths.) If the abscess is reached at an angle favorable for drainage, two courses are open—one to open and drain it completely at one sitting, and the other to do it in two stages, as in a lung abscess. If the latter course is decided upon, only as much pus is evacuated as is necessary to relieve extreme tension, and the needle withdrawn. The opening in the skull is then enlarged, the dura turned back and the exposed brain covered with cotton. The scalp is closed tightly over the cotton and left untouched for 24 hours, thus permitting a certain amount of walling off before the abscess is freely opened. At the second stage, which can usually be carried out without any anesthetic, the abscess is again opened and treated according to the desire of the operator, either by resection of the overlying brain, insertion of a Mosher drain, or simple drainage with a rubber tube. One must be guided in one's choice by the depth and position of the cavity and by the thickness of its wall. I believe that where it is applicable, the King operation should be of great value, though my experience with it has been small. I do not believe that one should resect the motor cortex, speech or visual centers, or brain in the vicinity of these structures in order to apply this procedure. If the surgeons prefers to handle an abscess at one sitting, rather than two, the same procedure can be carried out or a bone flap turned down if desired. The one outstanding disadvantage of the two-stage operation is the possibility of losing the abscess and not finding it again. This I believe is more than outweighed by the lessened risk of meningitis if this procedure is carried out.

We should remember that brain surgery demands a careful and delicate technique—one that differs markedly from any other kind of surgery and that we must apply this technique in our handling of brain abscess. Careful and accurate hemostasis is essential, and this cannot be obtained by hemostat and ligature alone. The use of Cushing clips, cotton pledgets and bits of muscle should be familiar to us all. More recently has come the use of the Bovee apparatus, or some modification of it, whereby cutting and hemostasis are obtained by means of electrocoagulation or desiccation. Such an apparatus is a great help in all brain surgery and almost an essential for excision of brain tissue, as in the King operation. I have spoken of the use of a Cushing ventricle needle to explore for abscess. For such use the hub of the needle should be bored to take a syringe point or, better, a metal adapter fitted to a rubber tube. If this is used pus need not be spilled in the wound. It is essential to keep

the needle in place after the abscess has been reached until the opening has been sufficiently enlarged and the drain inserted. The drain should project well into the abscess cavity and be held in place by sutures in the scalp. Personally, I have found the Mosher drain more satisfactory than any rubber tube or combination of tubes. Drainage of acute cerebellar abscess with the usually coexisting sinus infection is very definitely in the province of the otolaryngologist. Chronic cerebellar abscess should be drained through a clean field.

Everyone must use his own judgment in the after-care of brain abscess, and the process varies so much that each case is more or less a law unto itself. The essentials are that drainage shall be complete, that the drain shall remain undisturbed for a long time, weeks if necessary, and that the back wall of the abscess be left intact. I have never made any attempt to sterilize the abscess cavity. If there is any possibility of extrusion of the wall of a thick-walled abscess, it should be encouraged, but no attempt should be made at rapid removal. If there is a complicating meningitis, aseptic or septic, it should be treated by repeated lumbar or cisternal puncture.

The prognosis of brain abscess depends on the virulence of the organism, the position of the abscess, and the presence or absence of a so-called capsule. It is true that the early soft-walled abscess is difficult to treat and the results are not good. I do believe that there is little to be gained by precipitate operation on a septic infarct in the hope that it may be an abscess.

The neurosurgeon can bring you nothing new and startling in the diagnosis of brain abscess. He can perhaps offer you certain refinements in the technique of brain operations which are not needed in operations on the nose, throat and ear. He must, however, call on you for assistance in the care of the original focus in most cases of brain abscess, and it is only through the continuation and extension of the co-operation which has existed between the otolaryngologist, the ophthalmologist and the neurosurgeon that we may further benefit our patients and increase medical knowledge.

270 Commonwealth Avenue.

BRAIN ABSCESS. METHODS OF DRAINAGE.*

DR. HARRY P. CAHILL, Boston.

It is a bold task to address you on the subject of brain abscess when one is not bringing forward anything new or original. Were it not for the fact that I feel so earnestly the advantages of the transmastoid and subtemporal approaches in most cases of otitic brain abscess, I should hesitate to advance my views on this subject for your consideration.

Results in the treatment of brain abscess will vary greatly with the nature of the condition. No definite method will be suited to every case. Any procedure will demand a complicated technique. Any operation will be dangerously traumatic. The simpler we can make its execution, the more perfectly it will be carried out, and the evils of hemorrhage, edema and laceration of tissue will be greatly lessened.

This technique of necessity embraces two salient points: *1.* The avenue of approach; and *2.* the method of drainage. The simpler we can make this approach, the simpler our type of drainage, giving due consideration to the problem before us, the better our results must be.

In abscess of the temporosphenoidal lobe, the approach must be either through a bone flap, a temporoparietal decompression, a subtemporal decompression or through the mastoid tegmen extended. Approach by means of the bone flap gives a large field of exposure, some advantages if a certain type of meningeal abscess is present, and less danger of infection if the abscess is not located. But it does add to the operative time; it does add to the possibility of trauma. Hemorrhage from the posteroinferior border of the bone flap may be exceedingly troublesome. The abscess, if localized, is given opportunity to invade freshly opened tissue. Accidentally, even the infected aural area may enter the field. Theoretically, at times it has advantages; practically, in the hands of the otologist this is not so.

The subtemporal decompression has many of the favorable points present in the bone flap. In moribund cases with a sclerotic mastoid, not yet operated upon, it is approach of choice. When the mastoid is of a type requiring the use of a mallet and gouge, or the technique of the operator includes is as a necessity, this operation is of value.

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Approach through the mastoid or tympanic tegmen has given a higher average of recoveries than any other method; the meningeal approach and even the cerebral approach is often walled off. Here granulations on the dura or exposed dura guide one to the meningeal or intracerebral abscess in over 50 per cent of cases. Here, the abscess is frequently nearer the cortex. Here, a fistulous stalk opens at times. Here, the cortical cells do not belong to any important cerebral centre. In meningeal and cortical abscess, and where the abscess is not localized, its disadvantages can be reduced by remembering these possibilities in the operative technique.

The ideal treatment of a brain abscess would be to remove it *in toto*. This has been successfully accomplished; but the surgical shock, the encephalitis, the hemorrhage and danger of infection from the stalk, if present, or from the diagnostic puncture, or from rupture of the capsule, are insurmountable obstacles in the way of its success.

Partial removal of the wall of the abscess by unroofing it, with herniation, greatly decreases the dangers of the removal *in toto*. You are all familiar with the essentials of the King operation. It is one of the brilliant achievements in American surgery. The danger of recurrence due to partial collapse of the capsular wall of the abscess cavity or to retained infection and secretion of the capsular wall, is reduced to a minimum. The secretory capsular lining, whatever little remains, is replaced by granulation tissue. Drainage is not a serious problem.

In frontal lobe and in temporosphenoidal abscess, especially if it is on the right side, the King technique must be given ample consideration. In cases unsuccessfully drained through the mastoid, it has taken care of the recurrence.

The other methods of treatment vary from complete evacuation and inspection of the lining capsule through the encephaloscope to simple repeated punctures.

The type of drainage may range from the fenestrated metal or glass cylinder and wire gauze to rubber tubing and tissue. We must be open-minded and let the emergency guide our choice. All drainage is for the purpose of emptying the abscess cavity so that the intracranial pressure may obliterate it. Once the drainage tube is in place, various aids may be employed to assist the cerebral pressure, such as rotation of the head, gravity, lowering of the top of the table, suction, etc.

The removal of a cone of cerebral tissue reaching to the capsular wall by means of the radio knife and loop of Bovie has proved very satisfactory in several recent cases. It has permitted the wire gauze drain to be placed more accurately and with less trauma and

hemorrhage. Hence the use of the larger Mosher drain will become more common.

The formation of a fistulous tract about the drain to the abscess cavity in part makes the capsule extend to the surface of the brain. This approaches in a degree the unroofing method of King. Hence the drain, when in proper position, should not, ordinarily, be disturbed for from four to six weeks. It takes a considerable time for neuroglia tissue to form about the tube. After that time, the stay sutures may be incised and the brain permitted to expel the drain slowly.

Lipiodol injection has been of value in several of our cases. Its use is indicated in all drainage cases with persistent purulent discharge, headache or other questionable signs or symptoms. It has made clear in some cases that the drain was not in proper position and was outside the capsular wall. It has shown how easily recurrence can be favored by imperfect collapse of the wall. It has been, in a way, a measure of the abscess drainage.

There is in the school of the present day one group favoring larger and larger drainage openings, while another is using repeated puncture and suction. Rubber drains of fine calibre are favored by the latter group.

In the treatment of cerebellar abscess as in abscess of the middle fossa, there is some question as to the avenue of approach. If symptoms or signs of labyrinthine involvement preceded those of cerebellar infection, the paramastoid route through Trautmann's triangle, with at times obliteration of the lateral sinus, is the route of choice. When the abscess is secondary to sinus involvement, drainage through the inner sinus wall has been our customary procedure. If exploration of the mastoid cells posterior to the sinus reveals the presence of an extradural abscess, the area of protective meningeal adhesions should be used.

The importance of deciding which of these three avenues has a given area of meningeal protection may mean the difference between success and meningitis. Like so many of our operative points, we are indebted largely to Dr. Eagleton for this. The wisdom of bilateral occipital decompression in such cases is better left to the neurological surgeon.

The importance of measuring the spinal pressure in temporosphenoidal abscess, and of the lateral ventricle of the unaffected side in cerebellar cases immediately before the operation, must not be overlooked.

In conclusion, the mistakes one may make in certain intrapiarachnoid, cortical and unlocalized abscess will be more than compensated for by the value of the protecting meningeal adhesions and cortical

reaction over the avenue of infection from the mastoid. The radio knife and lipiodol injection have proved valuable. The simplicity of this technique with the Mosher drain will appeal to the otologist who does not possess the facilities nor the opportunities for keeping his neurological surgery at its best.

520 Commonwealth Avenue.

DISCUSSION OF SYMPOSIUM ON BRAIN ABSCESS.

DR. JOSEPH E. J. KING: Mr. Chairman, I thank you for the very courteous invitation to take part in the discussion of these most important papers, all of which I have thoroughly enjoyed.

I first want to take an adverse stand in regard to Dr. Eagleton's remarks relative to his own power of "observation," for if one reads his book, which he says is full of errors, one will find the most minute descriptions of pathology, just what one should see if he looked. His description of pathology in his book makes one feel very much as if he had attended the autopsy himself.

I shall confine my remarks to the treatment of brain abscess. The majority of cases which which we have had to deal were cerebral rather than cerebellar abscesses. Regarding the prognosis, I do not think there is very much difference between the results obtained in temporosphenoidal and frontal abscesses. So far as the type of infection is concerned it has been my experience that in those in which the streptococcus is present in encapsulated abscesses, the results are more favorable. Next comes the staphylococcus, and the least favorable is the pneumococcus.

We have all seen cases of suspected brain abscess in which we did not know whether an abscess was present or not. In all such cases it is important to get rid of the source of infection first—except in the case of a metastatic abscess—either by a proper mastoid procedure, or a proper operation for ethmoid or frontal sinus infection. Much to my sorrow I operated upon a man for frontal lobe abscess in which the abscess was not present. He had frontal sinus disease, with an extradural abscess, and previous to the operation on his frontal sinus I made an unnecessary exploration in the frontal lobe. I had two cases which should have recovered, but which died, the one in which the frontal sinus operation was not done until too late, and the other in which the operation on the frontal sinus had previously been done three months before, but in which reinfection took place. After the brain abscess was practically well she developed a secondary brain abscess from the frontal sinus. In cases of doubt it is my opinion that one should first attack the source of infection, rather than exploring the brain and looking for something which may not exist.

From the practical standpoint, all we care about in any of these operations or procedures is that the patient lives. We do not care whether he survives as a result of one procedure or another. The procedure which gives the best results in the hands of the operator should be used. No one procedure will cure all brain abscesses, and I do not think that all combined will do so. There are certain cases in which the Mosher drain is probably the most logical one to use. There are some cases in which a small drain, especially in the deep-seated abscess, will probably do much better. Such a drain has been used by Dr. Coleman, of Richmond; and there are cases in which the procedure which I have suggested will probably give the best results. I do not think that any one type of operation will give good results in all cases. Therefore, we should be prepared to use the type of procedure applicable to the case in hand. Dr. Eagleton has used an inverted osteoplastic flap. Dr. Adson has used an osteoplastic flap with the drain brought out through its central portion. Dr. Dandy proposed a tapping of the abscess without aspiration to relieve the pressure, and let it go at that. Some claim that the contents of the abscess will become less and less virulent, and may even become absorbed, and state that the patient gets well. I do not know about this type of procedure. Many of these patients undoubtedly go home and are well for some time, but I am under the impression that it is not logical to let a patient go around with such a potential volcano in his head. Dr. Coleman has used a small drain, which is left in

position in the cavity for a long time, and it is surprising to know the results which he has obtained. In the group of cases which he reported to me there are included 20 cerebral and four cerebellar abscesses. Metastatic abscesses were not included. Of the cerebral abscesses, three died, two of meningitis before operation. Of the cerebellar abscesses two died. This is indeed an extraordinary group of cases.

I have advised herniation of those abscess cavities which are suitable for such a procedure after one case, in 1920, which accidentally herniated and recovered. Other cases which had been drained with rubber tubes have died. After three cases had recovered by allowing the abscess cavity to herniate, I was advised by Dr. Berry to write a paper on the subject. The procedure which I advised was described in this paper. In these cases and subsequent ones, herniation of the abscess cavity outside of the cranial cavity was desired and accomplished. These, and others later in which a similar procedure was done, recovered. I would not use this procedure in all cases. One cannot expect an abscess at the base of the brain to be cured by herniation. I think probably I have made two mistakes in the original procedure. In the first place, I think the opening in the skull should be somewhat smaller. In the second place, too much herniation should not be allowed. Practically all of the cases which I have had that recovered were encapsulated. When one inserts the brain cannula for exploratory purposes and the "capsule" of the abscess is located, there is a distinct resistance offered to the cannula, producing a certain sensation or feeling—similar to the resistance offered by a thin-walled rubber ball in mush. So, instead of making such a large opening in the skull and opening the abscess immediately, in the next case which I operate upon I propose to remove the overlying brain tissue and allow the abscess which has not yet been punctured to herniate somewhat, so that adhesions may form about it previous to opening on the following day, thus preventing meningitis. This suggestion is similar to that of Dr. Mixter. At the end of 24 hours a certain amount of herniation will have taken place, and adhesions will have formed between the brain cortex and the dura so that there is practically no danger of spreading the infection. Opening the abscess on the following day is nothing more than a dressing. The overlying brain tissue is of but very little value. This tissue bleeds but little when removed. Most of the cases with which we have to deal are in the temporosphenoidal or frontal regions, and but few in the parietal, so I doubt if it makes any difference whether this brain tissue which has lost its blood supply is removed or not.

While coming here yesterday I considered the question of overherniation of the abscess cavity, and thought that perhaps in some of my cases too much tissue was allowed to herniate. I was considering the idea of having some device made of similar material to that used in Dr. Mosher's drain, but rather the shape of an old cotton basket than like a cornucopia, of different sizes to fit the different sized abscesses. I considered inserting this type of basket-like drain into the abscess cavity, allow the cavity to herniate until it was on a level with the skull, then remove the device and prevent further herniation. Once the cavity has herniated until none of it remains in the skull, the object of herniation has been accomplished. From this point onward, the area should be allowed to granulate over and heal. This I propose to do with the next case of encapsulated abscess upon which I operate.

I have had seven cases which got well, and I have seen two cases die which should not have died. I have had several cases upon which I operated die, but in these cases no hopes for recovery existed. I want to reiterate the importance of late operation for brain abscess. Some patients may die by waiting, but there is no doubt that when encapsulation has taken place the prognosis is more favorable. In all cases in which encapsulation has taken place the patient has passed through a stormy period at first, and finally encapsulation took place. If we could only keep our fingers off, and not allow ourselves to be rushed into a situation due to the family wanting us to do something for the patient, and if we could wait long enough to allow encapsulation to take place, I think we will save many more of these patients. Only one of my cases which recovered was nonencapsulated.

Several years ago brain abscess by the profession in general was considered a hopeless situation as most cases did not recover. Therefore interest lagged.

In recent years, however, more and more papers have been written on the subject, and everybody has become more interested in it. So if at such meetings as these everyone tells everyone else what they think about it, and discuss the subject, eventually we will have improved methods and procedures, and in the end we will be able to lower the mortality rate in brain abscess.

DR. S. J. KOPETZKY: After the excellent symposium presented this morning, little remains for me but to summarize, to stress some of the salient points and mention some of the outstanding facts.

The standpoint of the ophthalmologist, of the surgeon and of the otolaryngologist have been presented to you. Let us look at the question from the standpoint of the clinician. As one sits amazed and stunned, listening to Dr. Eagleton, one realizes that diagnosis is still an art and cannot be done by any set rules or definite formula. And on that art hinges the handling of brain abscess. As Dr. King said, all the procedures combined and applied to certain cases would not cure them. We should first separate the cases in which we can reasonably expect a cure from those in which no method that we have on hand can produce a cure; and we should not operate on the latter cases. Such operations only throw surgical procedures into disrepute.

Dr. Eagleton stressed the history. We should know the pathology that preceded the suspected brain abscess, know it very definitely. Abscesses are either capsulated or noncapsulated, and I partly disagree with Dr. King that in all abscesses you should wait indefinitely for encapsulation. Abscesses that are of hemorrhagic origin, or come from infarcts into the brain, move too fast, the lesion develops so quietly that the patient is soon dead—before any evidence is presented that we can see clinically. So while it is proper to wait for rational development and not rush in during the very early period, still we must realize that certain abscesses will never form a capsule, especially in cases where the abscesses are multiple.

I wish there was agreement among the ophthalmologists. I wish the reports that we get were written in common terms that all could understand. One hears quite a distinguished member of this Society say that there is considerable confusion as to what a given man means by optic neuritis and swelling of the nerve head. That ought to be straightened out. We are not speaking the same language and it leaves us at a disadvantage.

The ear presents the definite lesion. The chronic cases produce an abscess of one type, and the acute cases another, according to the lesion in the ear. My own experience is that the average type produces an abscess close to the ear. With the hemorrhagic type of mastoid—the septic type—the lesion in the brain may be anywhere.

The neurologist's standpoint was not presented this morning. My experience with these gentlemen is that they are so impressed with the slow growth of tumors that as a class they do not appreciate the more rapid growth of abscesses and the swifter change in the picture in these cases. Usually the otologist will make the diagnosis before the neurologists have summarized the varied reflexes or absence of function in the nerves they study. We must learn to ascribe greater significance to minute changes in the nerve reactions, so as to get an earlier diagnosis localizing the given lesion.

There is much in what Dr. Eagleton says, and I would be the last to deny it. But men of Dr. Eagleton's genius are beyond the rules and regulations that govern the rest of us. Many of the observations he makes and speaks about, I hope later to be able to place in their proper category and use. I trust his observations, but I fear I am unable to follow his reasoning. The fault may be mine.

We are dealing this morning with abscesses and not with infarcts. That is an entirely different lesion. An infarct does not necessarily result in an abscess.

I would like to make one point. That is, that I thoroughly agree with Dr. Mixter that lumbar puncture can be done if performed as he describes it, in almost every case. It should not be done roughly and uncontrolled by a manometer.

It is hard to do justice to a discussion such as this in the time permitted, but I thank the officers for the courtesy extended to me to add what I can to the discussion of this difficult subject.

NASAL SINUS DISEASE IN SMALL CHILDREN.*†

DR. L. W. DEAN, St. Louis.

Gentlemen: It is an honor and a pleasure to appear before this scientific body. I expect to be greatly benefited, not only from the discussion which I hope will follow this paper, but in presenting this subject to you. I must confess that to me many of the fundamentals of nasal sinus infection are very obscure. Little is known of the physiology of the sinuses; our knowledge of the action of the cilia in the sinuses is indefinite; I know but little of the action of the drugs we use upon the ciliated cells or of the influence of dust, of smoke, of temperature changes, or of humidity upon them.

Our knowledge of the fundamental changes in the lining of the sinuses which allow infection to take place is so limited that the whole subject is obscured. The work of Daniels at Iowa, of Wolbach at Harvard, of Arthur H. Smith at Yale, of Shurly at Detroit, and of Wenner at Washington University, suggest that we must look beyond the presence of the infectious organism to ascertain the real cause of infection.

When Shurly showed that the degree of infection was determined by the virulence of the bacterial organism plus the resistance of the cell, he did much to clarify the situation.

Laryngology today is making great progress because laryngologists are internists with special laryngologic training. We not only consider the influence of laryngologic conditions upon the organism as a whole, but we are particularly concerned with the influence of systemic disturbances upon the nose and throat. The latter is doing much for our patients.

No discussion of this subject would be complete without a consideration of the etiological factors concerned. In children, because the chronicity is not so great, they are not so difficult to discover; they are, however, numerous and complicated. We must think of the following things: Deficient diet; poor hygiene, including improper clothing and ventilation; allergy; metabolic disturbances; climatic conditions: especially does lack of sunlight influence its production; swimming; endocrine disturbances; nephrosis; diseased ton-

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sils and adenoids; nasal blockage; infection, especially in contagious disease.

The ease with which infections invade the mucous membrane is dependent on one or more of the predisposing factors. In the absence of allergy, it is evident that a perfectly healthy robust child not only resists infection better than a poorly nourished child, but when infection is present, it does not have the same clinical significance as in the former. In the robust child it may be of little importance and can readily be eradicated, while in the poorly nourished child it may exert a deleterious influence and be difficult to cure. It is impossible to consider sinus disease in infants and young children without considering deficient diets as important etiological factors. It is more important to know what prepares the tissues for infection than to know the infecting organism. If it were not for allergic states, nutritional disorders, and endocrine disturbances, we would not have many cases of sinus disease.

Dr. Daniels showed that every white rat fed on a diet deficient in Vitamin A developed a sinus suppuration. The control rat, living under the same conditions but with a normal diet, had unchanged sinuses. It was also shown that in the early stages of sinus disease the proper diet would control the sinus disturbance. As the nasal sinus disease became chronic, it could not be cured by diet. The natural conclusion is that while a diet rich in Vitamin A is an essential thing in the treatment of nasal sinus disease, it does not take the place of proper laryngologic treatment. This must accompany the dietary therapy.

Perhaps vitamins and proper hygienic measures play a more important role in our climate than in the tropics. Dr. Lambert, in a personal communication, told me that in Porto Rico a rickets commission found almost 100 per cent malnutrition in children, but only two cases of rickets.

Diets deficient in vitamins plus something else cause sinus infection. Shurly has shown that Gram negative organisms in the nose and throat other than the meningococcus, as the Micrococcus catarrhalis, become pathogenic when injected into animals fed on diets deficient in Vitamin A. He further showed that changing the biochemistry often changes the course of the disease. He mentioned the seasonal variation in the virulence of the bacterial flora of the upper respiratory tract.

From the time we are born until we die we combat the streptococcus. Early in life he is in the lymphoid tissue of the throat, later in the sinuses, then in the gall bladder, the teeth, the appendix, then

he lives in the prostate, the cervix of the uterus, in the colon. It is impossible to cut out all of the places in which he lives. By keeping up our immunity, our resistance, or what not, we fight our battle best. Vitamins and proper hygienic measures are the important things in this battle.

Wolbach and Howe, *Journal of Experimental Medicine*, 1925, studied the influence of diet deficient in Vitamin A on white rats. They found that the specific changes which follow the deprivation of Vitamin A in white rats concerned the epithelial tissue. This effect is the substitution of stratified keratinizing epithelium for normal epithelium in various parts of the respiratory tract and in some other parts of the body. They showed that the replacement of epithelium arises from focal proliferation of the cells coming from the original epithelium and not from change of pre-existing cells. There is an increase in the growth activity of the epithelium. They have definitely shown that this change is not secondary to infection, but is presumably a primary effect of the withdrawal of factors essential for the chemical activities or maintenance of differentiation of epithelium concerned.

Wolbach and Howe, *American Journal of Pathology*, 1929, also showed that the influence of Vitamin A deficiency in the guinea pig produced the same changes as were produced in the rat. That is, there is a hyperplasia and a keratinization of the epithelium. It is most important to know that these changes preceded the infection.

Tyson and Smith, *American Journal of Pathology*, 1929, found that the principal changes associated with a Vitamin A deficiency in rats are a metaplasia of the cuboidal or columnar epithelium in certain parts of the body, epithelial hyperplasia, and infection. They found that if the results of the dietary deficiency are not too severe, certain of these changes cleared up rapidly with the administration of cod liver oil. The extent of healing depended largely upon the destruction that was due to the infection that was present; that the infection persisted after the administration of cod liver oil. Perhaps we may feel that the proper diet will give a satisfactory result in sinus disease if it is administered before infection enters the picture.

The fact that these changes in the epithelium appear before the infection is very instructive. It is important if we are studying the etiology of sinus disease.

Dr. Wenner, working at Washington University, has, in at least one instance, noted the disturbance of calcium metabolism of the cells which preceded the hyperplasia and the keratinization. He has not confirmed this observation, consequently it has no significance.

These experimentors often mention a metaplasia of the epithelial cell. Personally, I doubt if there is such a thing as true metaplasia in the epithelium of the sinuses. What we have is a change in the shape of the cell due to the pressure of a rapidly growing epithelium.

The allergic nose must be watched for in children. Many of these allergic patients have unnecessary operations. Just before I left St. Louis, Dr. Eyermann observed a patient with an allergic nose. Fifty operations had been performed on the nose without benefit. I am sure that we can all imagine the result. Many patients have been seen who have had 25 operations.

There is no objection to operating the allergic nose in children if they need operating. Many have a suppurative sinusitis in addition to the allergic thing. But to operate for the allergic condition which will disappear with treatment is positively not justifiable. To illustrate: A boy, age 11 years, had a sinusitis with white discharge coming from both nostrils. The trouble came on in July without history of exposure to inclement weather. He had had the same thing before. Extensive operations on the sinuses had been advised. He had 40 per cent eosinophils in his nasal discharge with only an occasional polynuclear cell. The maxillary sinuses especially were blurred. I insisted that he have only allergic treatment and he completely recovered. I will show slides of this case later.

Another case came under my observation. The maxillary sinuses were blurred on X-ray. He had been advised to have a Caldwell-Luc operation but refused. He had no treatment; in a few weeks his sinus plates were clear.

Dr. Proetz and I have a patient with a typical history of an allergic nose. Amost careful search failed to reveal any evidence of sinus disease or other nasal pathology. Lipiodol was placed in the maxillary sinuses on Wednesday. There was no thickening of the lining membrane. On Thursday night the patient had a little chest discomfort suggestive of asthma, but no subjective nasal disturbance. On Friday the sinuses were again X-rayed. The sinus lining was enormously swollen and most of the lipiodol had been forced into the nose.

Just before leaving St. Louis, I saw a patient with recurring nasal discharge of 15 years' duration. No one had found any evidence of sinus trouble. His nose reveals the characteristic edema of allergy. He has 60 per cent of eosinophils in his nasal discharge. I told him that all of his treatment is to be given by an internist. To me an eosinophilia of 10 per cent or more in the nasal discharge indicates allergy. My reason for this is that I have found no exceptions.

I do not know the significance of eosinophilia. The absence of eosinophils in the nasal discharge does not exclude the allergic nose.

Dr. Wenner, who is research physiologist in our department, has made a most interesting observation regarding the experimental production of eosinophilia in a laboratory animal. The animal used in his investigation was an albino rabbit which Dr. Nemours, the comparative anatomist on our staff, has found to lend itself very favorably to maxillary sinus experimentation.

The method used consisted of injecting a solution of sodium alizarin sulfonate by an external route into the maxillary sinus of the experimental rabbits. A series of control animals run simultaneously with the experimental received injections of distilled water.

Immediately following the administration of alizarin, the cilia of the epithelial cells became paralyzed. The ciliated epithelium from the buccal cavity of the frog show a similar reaction to this substance. Histological sections of the sinus epithelium of rabbits treated with alizarin show infiltration of eosinophils into the basement membrane and emigration into the sinus passing between the epithelial cells. In addition the surrounding maxillary bone showed many areas of bone absorption quite similar to that found in allergy.

Sodium alizarin sulfonate is a dye having wide commercial, chemical and pharmacological usages. Its main pharmacological action consists in its ability to precipitate calcium and related inorganic elements; *i. e.*, strontium and barium. With calcium, calcium alizarinate, a red precipitate is formed. Dr. Pollack, of Cornell University injected alizartin by the Chambers' microdissection method into *amoebae dubia*. Immediately pseudopodia formation ceased; the red calcium alizarinate granules could be seen diffusely scattered throughout the cytoplasm, thus actually locating the calcium ions in the protoplasm. Fuehner, in Germany, reports that alizarin interferes with the motility of the phagocytes.

Since alizarin itself is nontoxic, the main eeffcts are due to a change in the ionic ratio of the tissues affected. The precipitate of the calcium ion from the cell destroys cellular equilibrium, thus favoring increased permeability, changes in electric potential and osmosis.

Mr. Linton in following up the bacteriological side of this problem has found that after stagnation, infection immediately ensues. His *in vitro* experiments demonstrate that excised sinus epithelium show very significant chemotropic and thermotropic responses. Thus ciliary action may be kept for a number of days if kept at a temperature of 4° C.

Mr. Linton concludes from his preliminary work that bacteriostatic properties of the secretions of the sinus epithelium is a factor *secondary* to ciliary action in maintaining sterility.

These observations of Dr. Wenner and Mr. Linton are not presented here in their entirety, nor are they presented as being of any immediate importance. They are, however, a significant part of a carefully developed plan of research on the nasal sinuses. Further experiments are under way.

I have not been able to observe the influence of ciliary dysfunction in the sinuses upon stagnation and infection except in the case of the rabbit studied by Dr. Wenner. Dr. Proetz and Dr. Johnston have devised an apparatus which will give illumination of and opportunity to study the interior of sinuses in animals through windows. The research which they have planned will, I am sure, add to our knowledge of this subject.

I am inclined to think that the allergy does interfere with ciliary action and cause stagnation in the sinuses with resulting infection. The infection once firmly established may indicate the need of an operation on the sinus. When discussing asthma, I will mention a type of allergy in which I believe operation on the sinuses for drainage and ventilation is indicated.

Perhaps I should mention here that the allergic nose is often benefited temporarily by any kind of a nasal operation, even by washing out the maxillary sinuses when the washings are perfectly clear.

The discussion of asthma always brings in the question of allergy and it may simplify matters if we discuss it now. Asthma in children is often benefited by treating a nasal sinusitis. I have referred to a pediatrician a number of such cases at least a year after treatment and this observation has been confirmed.

Most of the cases where work on the sinuses has been done have been those where the pediatrician has not been able to discover an allergen that is associated with the condition. The patients that we treat are those who have polynuclear leukocytes in the discharge from the sinuses. They are patients with a history of an upper respiratory upset called a cold, or what not, accompanied by asthmatic attacks. Vaccines and allergic treatments do not give a satisfactory result. There is evidence of sinus disease and the polynuclear cells are in the discharge during the acute attack. Washing the maxillary sinuses by needle puncture gives temporary relief. I realize that the traumatism of such a procedure may produce this result. It is the constant appearance of evidence of infection in the sinuses at the time of the asthmatic attacks that allows one to predict a satisfactory result if the sinusitis is eradicated.

I do not know if there is such a thing as bacterial allergy. It has never been proven. Clinically it is reasonable to suppose that such a condition exists.

The following case is instructive. A patient, age about 9 years, had a diagnosis of tuberculosis of the lung with asthma. Dr. Marriott diagnosed a sinus lung and transferred the boy to our service. He had a bilateral maxillary sinusitis with no eosinophils in the discharge, but many polynuculear leukocytes. For one year he was treated. Washing the maxillary sinuses by needle puncture always relieved his asthma. When ventilation of the sinuses was recommended, it was refused and vaccines were used. After two months the father asked me where to take the boy for the winter because of his asthma. My answer was, "Why take him any place when ventilation of the sinuses will give a good result both for his sinus trouble and his asthma?" His asthmatic attacks were always preceded by an acute exacerbation of his upper respiratory infection. Three months ago windows were made in each maxillary sinus. The result is most satisfactory.

Malnutrition chronic in children is often associated with a chronic nasal sinus disease. Which condition is primary is often open to question. However, proper treatment of the sinuses is an essential part of the program. It often gives excellent results so far as the systemic condition is concerned.

Dr. Marriott often, after the study of a child with a diagnosis of chronic pulmonary tuberculosis, states that it is a case of nasal sinus disease. So far his observations have always been correct.

When I was associated with a sanitarium for tuberculosis, the pulmonary expert referred to us many cases of pulmonary conditions simulating chronic tuberculosis that were improved by the eradication of a chronic nasal sinusitis.

Pediatricians find a large number of cases diagnosed as tuberculosis and suffering from foci of infection. The primary focus outside the lung is in the head and often in the nasal sinuses.

As to whether the lungs are infected by the aspiration of infective material or through the lymphatics, or both, is still a question for debate. I doubt if all the laryngologists appreciate this close relationship between the nasal sinuses and the lungs.

It is a wonderful thing when parents, who have a diagnosis of tuberculosis made on their child and have been advised to seek a favorable climate, find that after months of treatment of the nasal sinuses, the condition has improved and that the disease is not tuberculosis.

Without discussing the relationship between acute infectious arthritis and infections about the nose and throat, we can say that while the sinuses are second to tonsils in the role of foci of infection, nevertheless they play a very prominent part. Many cases of acute infectious arthritis improve with the removal of tonsils and are only controlled by the treatment of a coexistent nasal sinus infection. Many cases without tonsils have the sinuses as the sole focus. Here we meet with the difficulty of successfully combating the sinus infection without making the arthritis worse. In winter with a history of an acute sinus infection and an acute joint appearing with it, it is exceedingly difficult to tell just what should be done if the sinusitis does not yield to simple treatment. Seeking a proper climate is, I believe, the best procedure for the time being, followed by active sinus therapy later if necessary. This is only indicated when progress is being made and the arthritis is improving. It may be necessary to drain the infected sinus right away. We must be guided by the combined judgment of the pediatrician and laryngologist in each case.

With all patients with systemic disease and sinus infection, the pediatrician should not only always be consulted, but I would consider his judgment better than mine. Before giving his opinion he should be informed as to the laryngologic condition, and the prospect of satisfactory results so far as the sinus disease is concerned by this or that treatment.

Chronic arthritis, or Still's disease, must be mentioned. In the cases of Still's disease that I have seen, there has been a very large percentage of cases of chronic suppurative sinus disease. In the opinion of some very excellent pediatricians, the result of the eradication of the sinus disease has been very beneficial. The patient has not only been benefited, but there has been a decrease in the acute joint manifestations. There is such a diversity of opinion as to the ultimate benefit to the patient that I think we should be careful in assuming that much can be accomplished.

As soon as I am back in St. Louis, we will study and work carefully on four of these cases. They will be observed carefully by Dr. Marriott and the result carefully evaluated. These cases vary so much in their clinical course, they get better, then worse, without apparent cause, that it is difficult to state that any change for the better following any treatment is the result of the treatment. The difficulty in our treatment will be the complete obliteration of the nasal sinus disease. If we are successful in this, I am very hopeful as to results.

Following colds or influenza there is often a condition characterized by fever, leukocytosis slight, and often malnutrition. The child does not do well. There is no nasal discharge. Examination usually reveals a nasal sinus infection. Until recently I thought all such children with nasal sinus disease had a cervical adenitis, but this is not the case. Even the nose may show no distinct evidence of disease of the sinuses. I think every child with an upper respiratory disturbance, followed by fever in which there is no demonstrable ear or lung condition responsible, should be considered as a probable residual sinus case. Treatment of the sinus condition gives a satisfactory result. If in such a case there is anterior cervical adenitis, with no tonsillar disturbance, one should consider that the trouble is probably in the nasal sinuses. At any rate, the trouble is somewhere in or about the upper respiratory tract.

It is often irritating to have the pediatrician send such patients to us with the statement that the treatment is wholly laryngologic. However, in my experience often when the only indications of sinus disease were fever and cervical adenitis, the pediatrician has always been right.

In nephrosis or parenchematous nephritis, as a part of the general edema we get a blurring of the nasal sinuses on X-ray, particularly of the maxillary. This is a result of the disease and not a cause. In the early stages, at least, there is no infection and the picture has no significance. Later, if infection is superimposed as the result of the edema and interference with ciliary action resulting in stagnation, the use of ephedrin often gives a good result because it shrinks the edematous membranes about the ostia so completely. Needle puncture and all procedures resulting in traumatism are markedly contraindicated because of the ease with which systemic infection is produced in this class of cases. Briefly, I think in most cases no treatment of the nose is indicated. The most that should be done is to use ephedrin.

Glomerular nephritis presents quite a different problem. It is associated with the streptococcus somewhere in the body. Occasionally, when the tonsils and adenoids are out, it may be in the nasal sinuses. When there is infection of the nasal sinuses and acute glomerular nephritis, the management of the nasal sinus condition requires that the most careful clinical judgment be exercised. Certainly an operation on the sinuses of any magnitude may produce death in a few days. When the streptococcus is in the tonsils we can take out the infected material and throw it away. We cannot remove all of the infection in the sinuses quickly by any procedure,

and we do not often get in sinus infections and nephritis, the brilliant results observed immediately following tonsillectomies. When the infection is in the tonsils we often see the blood disappear from the urine within 24 hours following the operation.

With nephritis and suppurative sinus disease where there is a history of upper respiratory disturbance and the nephritis appearing during the attack, if the tonsils have been removed it is essential that the sinusitis be treated. In the beginning the most conservative procedure should be used. Then, if necessary, we go to the intranasal surgical procedures as seems best.

Only very rarely is a radical procedure indicated. It must be done only after the nephritis has been controlled in so far as possible by the treatment of and intranasal operation upon the infected nasal sinuses.

The pathology of nasal sinusitis in young children differs from that of the adult, in that the changes due to great chronicity are not so marked. We find in chronic ethmoiditis the lamellae of the bone are roughened in cross-section, due to bone absorption. We find in the sinuses thickened periosteum, subperiosteal abscesses, mucosa much thickened and infiltrated with inflammatory cells. Cysts are found in the glands. Cilia are destroyed. So-called metaplasia, which I believe is nothing more than distortion of columnar cells because of the rapid growth and pressure, is observed. Edematous membrane with polyp formation may be present. Granulation tissue is common.

In the bone absorption abscesses are found in the bone. Fibrous tissue containing blood vessels or eburnated scarred bone may be present.

The allergic cases present the same pathology as in the adult. There may be hyperplasia of the epithelial cells; edema of the subepithelial tissue with an increase in the number of eosinophils present. There may be a round celled and lymphoid celled infiltration.

The first step in the treatment of nasal sinus disease in young children is the eradication of the cause. Only by taking into consideration diet, allergy, endocrine disturbances and all the systemic etiologic factors above mentioned can we possibly treat the condition. The securing of a painstaking history is of greatest importance.

The second step in every case, I believe, is the examination by the pediatrist. We must have a complete report as to the metabolism of the child, as to endocrine disturbances, as to allergy, and all things which would have any bearing on the condition. Proper diet must be prescribed; the ventilation of the house and the influence of cli-

mate, must be considered; clothing must be regulated; allergy, if present, must be controlled. The eradication of the predisposing factors, pediatric and laryngologic, together with the most simple laryngologic treatment, will control satisfactorily nine-tenths of all cases of chronic nasal sinusitis in children without any operative work on the nasal sinuses. In the course of a few years with constant attention directed to the exclusion of the etiological factors, the sinus disease will be cured.

It is quite essential that these children should play, swim and do all the things that normal children should do. There is no reason why they should not swim if the usual precautions as to the length of time that should be devoted to swimming, keeping the head out of water, etc., are observed. It is most essential that at least during the summer months the children lead an out-of-door life. After such a summer they are usually better so far as their sinus trouble is concerned, and each year they go through the winter with less trouble until a final satisfactory result is obtained.

Diseased tonsils and adenoids, if present, should be removed. Only rarely is it necessary to operate a septum. Deflected septa which seem to cause trouble if not operated in time have little significance. With the decrease in the pathology of the turbinates from the control of the sinus infection and the widening of the nose from growth, sufficient space for proper nasal function is present.

The ordinary treatment for acute sinusitis is indicated during the acute exacerbation. One per cent ephedrin is especially beneficial at this time. Occasionally in the chronic condition it is necessary to make a meatal opening in the maxillary sinus which will remain open and drain for a few months.

When polyps are present an allergic basis should be carefully sought. The polyps should be removed in the usual way. If they return after removal the cauterization with trichloracetic acid or other simple measures or the use of radium should be considered. Occasionally with sinus infection in children there is an apron of lymphoid tissue on the wall of the pharynx which persists in spite of sinus treatment. This should be attacked with X-ray.

An operation on the ethmoid labyrinth should never be done unless it is definitely demonstrated that diseased bone is present and a serious complication is proven to be due to the sinus suppuration.

Operations on the nasal sinuses of children other than meatal drainage of the maxillary sinuses is very *rarely* indicated.

Washington University School of Medicine.

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Hutter, in the Wiener Klinische Wochensh., Nov. 28, 1929, discusses the use of spleen diet in tuberculosis of the larynx. Good results were obtained by spleen diet in cases of tuberculous fistulas of the joints. This prompted Hutter to attempt the therapy in tuberculosis of the upper air passages. He gives a detailed report of one case which showed marked improvement. He later used the diet in the treatment of three other patients and obtained favorable results in two of them. He has several other patients under observation at the present time and makes the plea that other workers attempt to gather more information on this particular method of treatment.

Halle, in the Zeitschrft. fur Hals, Nasen u. Ohrenheilk., 25, 267, 1930, discussed the dangers from poorly devised instruments in the use of surgical diathermy. Having seen numerous poor results and trauma from poorly insulated instruments, he decided to devise a laryngological instrument which should, as far as possible, do away with this possibility. All metal parts are buried deep in hard rubber, so that when the connections become moist there is still no diversion of the current to undesired places. With instruments made according to this plan, a minimum of bad results was obtained from this cause. KELEMEN.

Glenn and McGinnis, in the Journal Missouri State Medical Association, January, 1930, report the use of water-cooled mercury quartz lamp with a laryngeal applicator in the treatment of 77 cases of laryngeal tuberculosis. Their strongest point is that the treatment is justified if there is no other reason than that it affords symptomatic relief.

Kraus and Wirkner, in the Zeitschrft. fur Hals, Nasen u. Ohrenheilk., 25, 270, 1930, present an article entitled, "Anatomical and Roentgenological Examinations of the Mastoid Emissary Vessels." They examined 100 skulls and agreed with the previous knowledge that sinus anomalies are relatively rare. The connection through the emissary vein is independent of the size, course and position of the sinuses. Emissary veins occasionally may carry blood back to the posterior fossa. The upper knee of the sinus is the place of predilection for the union to the outside. One finds that anomalies of the emissary vein go hand in hand with lack of pneumatization of the mastoid process, and, in a total lack of pneumatization, one finds very narrow and small vessel unions and many accessory vessels. KELEMEN.

Gordon and Litvak, in the Medical Journal and Record, New York, Jan. 1, 1930, discuss the differential diagnosis between diphtheria and oral lesions of blood dyscrasias. They considered agranulocytic angina, acute lymphatic leukemia, myelogenous leukemia and aleukemic leukemia. They state that a complete blood study is indicated in every case of suspected diphtheria in the absence of any of the following: *1.* Persistence of membrane after the administration of diphtheria antitoxin; *2.* lesions in the mouth other than on the tonsils; *3.* cutaneous and mucous membrane hemorrhages; *4.* marked pallor; *5.* general glandular enlargement; *6.* enlargement of the spleen.

Shurly and Turner, of Detroit, in the Feb. 22, 1930, issue of the Journal American Medical Association, write on the Infection of the Accessory Sinuses and Upper Respiratory Tract in Vitamin A Deficiency. The work described is the result of a Research Grant by the Detroit Board of Education in their attempt to control acute infections among children of school age. The work done corresponds very closely to that reported by Daniel and Armstrong, of Iowa City, who placed 30 new-born infants in the pediatric ward for the first two years of their lives. Not one of these cases developed infection of the ear or sinuses, notwithstanding the prevalence of these infections in adjacent wards. One exception to the above statement was a case in which the mother, in preparing her child's food, skimmed off the surface of the milk (of the mixture of cod liver oil and milk feeding). These children are now 8 years of age and they all have perfect teeth at the present time. The only exception being a case of mastoidities in the child whose mother skimmed the cod liver oil from the milk. MYERS.

Else Levy, in the Zeitsch. fur Hals, Nasen u. Ohrenheilkunde, 25, 245, 1930, describes an interesting case of transient amaurosis during septum anesthesia. The usual procedure of ½ per cent novocain plus a drop of adrenalin to the c.c. was used, when the patient suddenly complained of severe pain in the eye and that he could not see. The left eye became completely amaurotic and the left pupil went into complete dilatation. In about 15 minutes the pupil began to react and a few minutes later operation was completed and the symptoms quickly disappeared. The explanation is given that in the injection into the mucous membrane some small branch of the ethmoidal arteries must have been entered. Pressure in these arteries is very slight and the injection must have come to the central artery of the retina. In the described case there was a rapid resorption but the author brings up the thought that in other cases where one used a substance such a paraffin then the resorption might not occur and a more or less permanent damage might result.

KELEMEN.

F. P. Herman, of West Palm Beach, Fla., in the February, 1930, American Journal of Surgery, discusses the use of tonsillar electro-enucleation versus tonsillar coagulation. The article is concise and beautifully illustrated to show the technique of electro-enucleation, and rather astounding results are recorded. Unfortunately, the author does not delve into the question of postoperative slough and hemorrhage, except for the brief statement that the postoperative result is usually bloodless.

Bigler, in the Zeisch. fur Hals, Nasen u. Ohrenhilk., 25, 249, 1930, writes on the X-ray demonstration of abscesses in the apex of the temporal bone in a Gradenigo's symptom complex. The picture was taken according to the Stenver technique and showed with extreme clearness a pathological process in the pyramid of the temporal bone.

KELEMEN.

Hollender, of Chicago, in the American Journal of Surgery, for February, 1930, presents a short article on the use of physical energies in oto-rhino-laryngology. He discusses the use of radiant heat, light, infra-red, ultra-violet, Galvanic current, zinc ionization and diathermy. He discusses the indications and uses of these various agents and in summing up is quite optimistic as to the benefits derived; in fact, much more so than contemporary literature on the subject would lead one to expect.

Caesar Hirsch, in the Zeitsch. fur Hals, Nasen u. Ohrenheilk., 25, 256, 1930, comments on the connection between Bang's Disease and Rhino-Laryngology. This disease is produced by bacillus abortus Bang. The patient is suddenly taken ill with headache, chills, marked sweating, often accompanied by sore throat. The complete picture greatly resembles Malta fever. It is of interest to Rhinologists, in that should a tonsillectomy be undertaken, one must be able to recognize the constitutional symptoms or this disease may be reawakened by the operation. KELEMEN.

O. Muck in the Zeisch. fur Hals, Nasen u. Ohrenheilk., 25, 262, 1930, describes a transitory hypacusis following caloric testing of the labyrinth. This phenomenon is observed in the syringed ear when cold water is used, but in the opposite ear when warm water is used. The author concludes that this is apparently caused by a cramp of the cochlear branch of the nerve. KELEMEN.

E. Watson-Williams, of Bristol, in the British Medical Journal, Dec. 14, 1929, reports 15 cases of mastoid disease without otorrhea. He found that there is often no preceding illness and that there is, or is not, or never has been pus from the middle ear and that pain ma ybe very slight, if present at all. There may be little or no tenderness and fever is a very unreliable sign, being absent in many cases. There may or may not be some edema over the mastoid, and the tympanic membrane usually appears quite normal. The otologist, despite all these negative findings, has to be on the lookout for a mastoiditis. Very frequently complications give the first warning of any deep pathology.

H. I. Lillie, of Rochester, Minn., in the Feb. 22, 1930, Journal American Medical Association, presents an article on "Otitis Media and Diseases of the Mastoid; Early Involvement of the Blood Stream." During a period of two years he observed nine cases of blood stream infection, many with positive blood culture, in which he did not find it necessary to disturb the jugular vein of the lateral sinus. He calls attention to another important fact,—that a diploic mastoid with hemorrhagic aspects is more dangerous than the confluent type; but makes a plea for blood transfusion before mastoidectomy. This is a departure from the generally accepted viewpoint as to ligate the jugular or to interfere with the sinus in the mastoid, but Lillie quotes recent authors as evidence. He does insist, however, that the mastoid be thoroughly cleaned and searched in such cases where there is a suspicion of sinus involvement. MYERS.

BOOK REVIEWS.

The Neck. A Roentgenological Study of the Soft Tissues; Consideration of the Normal and Pathological. By Percy D. Hay, Jr., Roentgenologist of McLeod Infirmary, Florence, S. C. Introduction by Henry K. Pancoast, M.D., Professor of Roentgenology, University of Pennsylvania, Philadelphia. Thirty-six Roentgen-Ray Studies. New York: Paul B. Hoeber, Inc., 1930.

This is indeed an unusual monograph. The book itself was inspired by the Jackson Bronchoscopic Clinic for examinations of disturbances in the lower portions of the neck, because, as everyone knows who has anything to do with bronchoscopic work, this region has become the *bete noir* of the work. Dr. Pancoast, who writes the foreword of this monograph, calls attention to the importance of Caldwell's position, in making the X-ray in these cases. Under proper conditions retropharyngeal abscess, retrotracheal abscess and other obscure conditions in this region can be diagnosed by the X-ray as readily as can the metallic foreign body.

In the reviewer's opinion, this book should be a part of the library of not only the laryngologist, but also the X-ray man by whom and with whom these cases must of necessity be collaborated. E. L. M.

Harelip and Cleft Palate; Cheiloschisis, Uranoschisis and Staphyloschisis. History, Etiology, Development, Anatomy, Physiology, Types, Surgical and Non-Surgical Treatment, and Reported Cases. By Matthew N. Federspiel, B.Sc., D.D.S., M.D., F.A.C.S., F.A.C.D.; Professor Oral Surgery, Marquette University; ex-Professor of Orthodontics, Marquette University; former President, American Society of Orthodontists, etc. With 156 illustrations by L. Kerschbaumer, M.D., St. Louis: C. V. Mosby Company, 3525 Pine boulevard. Price $5.00.

The opening chapters of the book deal with histology, anatomy and physiology of the face and head. Illustrations are clear and concise with the reading matter presented in a brief, interesting fashion. The types of harelip and cleft palate are then discussed. This is logically followed by the surgical treatment of each type. Methods of technique are clearly explained with liberal illustrations to demonstrate the essential points. There is a lengthy chapter on the non-surgical treatment in which the various protheses and appliances are described and illustrated.

The closing chapter deals with case reports with pictures showing the condition before and after treatment.

The clarity and conciseness of the book make it a valuable adjunct to the general surgeon's as well as the specialist's library. M. F.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Meeting of Nov. 8, 1929.

Mastoiditis with Perisinus and Extradural Abscess. Dr. Harry Neivert.

Mr. J. G., age 46 years, came to me five weeks ago with a history of having had a cold in the head nine weeks previous, which was followed by an acute otitis media, necessitating a myringotomy. He was treated during this time by a specialist, and apparently the progress was satisfactory. There were no untoward symptoms, except a thin purulent discharge with occasional vague pains in the right temporal region and tinnitus. At no time was there any fever.

On examination there was no tenderness anywhere in the mastoid region, the auditory canal contained pus, the tympanic membrane was thickened posteriorly with a nipple perforation in lower posterior quadrant. I enlarged the perforation and because of the improvement in the discharge and in the condition of the tympanic membrane, I continued treatment two weeks. Then, on account of increase in discharge, I had an X-ray examination made, which was reported negative. Three days later, I had another X-ray examination by another man, but before the plates were ready, the patient's wife notified me that Mr. G. was having a dizzy spell and vomited. I ordered him to the hospital, where I operated immediately.

I found a very large mastoid, cells in the zygoma, above the temporal crest and around the facial canal. There was no pus in any of the cells, only thickened lining granulations. On exposing the sinodural angle, where the cell walls were completely absorbed, I found a mass of granulations, with complete loss of the tegmen mastoidi and upper sinus plate. On probing, a gush of yellowish pus, about two or three teaspoonsful, came out under pressure. I did not disturb the granulations, but placed drains and left wound open without suturing. The postoperative course was again without a single rise in temperature or any other symptoms. Culture of pus showed no growth after 24 hours.

It is now three weeks since operation. The middle ear was dry immediately after operation. I am still keeping the wound partly open. This case is reported because the symptoms and signs gave no definite indication of the pathology found. Certain signs made me suspicious, but I did not feel justified in operating.

Mastoiditis, Complicated by Pregnancy and Lateral Thrombosis. Dr. Lee R. Pierce.

The following case report of mastiditis, complicated by pregnancy and lateral thrombosis, presents the following history and findings:

Mrs. D. K. contracted influenza during the first week of January, 1929. The following week a severe pain developed in the right ear and myringotomy was done the following day. The next day severe vomiting associated with dizziness occurred, which persisted for a week. At this time there was present a pregnancy of about four months' duration.

One week after the myringotomy was done it was found necessary to do a simple mastoidectomy, which immediately relieved the vomiting and dizziness, but the temperature continued to be elevated from two to three degrees. Two or three blood cultures taken at this time were returned negative. The patient was discharged three weeks after operation and returned to New York City. She states that at the time of her discharge the posterior wound had not healed and that there was considerable discharge from this area, as well as from the middle ear.

I saw the patient on March 15 in consultation with Dr. F. W. Splint. The symptoms were as follows: There had been a sensation of falling forward whenever the patient attempted to walk. No headaches or temperature. The

pregnancy was progressing favorably and the patient felt well, except for a loss of strength which she had not been able to regain.

Examination disclosed the following: Posterior wound open the entire length of incision and filled with infected granulations. There was an area of bone in the position of the posterior ridge, which was exposed and was dark in appearance. There was a fistulae leading into the zygomatic area, which was discharging. The middle ear showed a moderate-sized perforation of the drum posteriorly. The external canal was filled with pus, no tenderness along the course of the jugular vein. There was slight tenderness on pressure along the posterior edge of the mastoid wound.

Hearing Tests: Conversational voice and forks not heard in operated ear. Weber referred to unoperated ear, 1¹; left ear, normal hearing. Caloric test, right ear, no response. Caloric test, left ear, normal reaction. Examination of the fundi of both eyes was negative, the neurological findings were negative except for the tendency to fall forward. Hemoglobin, 65 per cent; red cells, 3,000,000; white count, 6,000. It was evident that the labyrinth of the operated ear was dead and that operative interference was necessary but it seemed advisable to wait for the termination of the pregnancy, which had progressed into the seventh month, and the patient would be under the care of a trained nurse and her physician.

I did not see the case again until after she had been taken to the hospital; this was during the second week of May. The following history was obtained: The patient had been as well as usual until one week before coming to the hospital, when there had been a severe chill, followed by a rise in temperature to 103° and severe right-sided headache. The following day jaundice developed. Two days later there was another chill, with sudden rise in temperature from 99° to 103°. Examinations at this time were made for malaria and were found negative, the jaundice improved, but the sudden rises in temperature persisted. It was then decided her condition would improve by inducing labor, which was done, May 3, and a normal child delivered. The symptoms improved considerably following delivery, the temperature remained normal, headaches disappeared and the patient appeared to be on the way to recovery, when she was suddenly taken with a chill with rise of temperature to 104° on the fourth day following delivery. I saw her the following day; she was having severe headache, pulse weak, rate, 120; temperature, 99°. The findings on examination of the left ear and the appearance of the posterior wound were the same as on the previous examination on March 15. Neurological findings were negative. There was no tenderness over the jugular, slight enlargement of lymph nodes in the right side of the neck along the course of the jugular vein. Eye grounds were negative. Blood cultures taken that afternoon and on the following day were negative; hemoglobin, 50 per cent; red cells, 3,000,000; white cells, 1,400. I felt that the diagnosis was most likely a lateral sinus involvement after examination of the temperaaure chart, especially as there were no symptoms of other complications. The medical men in attendance were not inclined to agree with me so Dr. W. C. Bowers was called in consultation and agreed that a lateral sinus involvement was present. Operative interference was immediately decided upon; the patient's general condition was not at all good, so a blood transfusion was given as soon as the patient was under anesthesia. Dr. Hugh Blackwell, who assisted at the operation, suggested that we expose the jugular vein first, which was done. It was found to be partly collapsed and contained clot. The vein was resected to the bulb and removed; the posterior mastoid wound was cleaned of all granulation and necrotic bone, the membranous sinus was exposed and found to be filled with clot and small pockets of pus. The clot was removed as far as possible toward both ends of the sinus, but we were unable to obtain free bleeding. The posterior wound was closed with drainage. The patient was in shock that night and the following day, necessitating another transfusion. There was a slight chill with rise of temperature to 103° the third day following operation, which was repeated again on the fifth day following operation. From this time on the recovery was uneventful. Five weeks after operation the neck and mastoid wounds were healed.

Examination of hearing in the operated ear during the latter part of July was negative for all tests. These tests were made with the noise apparatus

in the opposite ear. On examination again during September I was very much surprised to find that conversational voice was heard at about six inches and that the Weber was referred to the operated ear. The last examination two weeks ago showed conversational voice heard at 18 inches. Caloric test operated did not produce reaction.

DISCUSSION.

DR. KOPETZKY: The case presents so many unusual features that I can only congratulate the operator on the excellent result of the outcome, and the establishment of the diagnosis on a dead labyrinth, which later was found to have recovered function. That is rather an unusual event.

DR. JOHN GUTTMAN: The reader of the paper stated that the patient did not show any reaction to the caloric test. Was there any other test like the turning test tried? If the labyrinth on that side was dead, how was it that the patient had some hearing on that side? We know that in a destruction of a labyrinth by an acute suppuration the cochlear nerve being more sensitive, becomes destroyed first. I did not hear it mentioned that there were any symptoms of an acute inflammatory or suppurative labyrinthitis.

DR. DWYER: It is certainly very unusual to have dead labyrinth and then to get hearing, but I have always felt that we went too far in labyrinth disease in declaring definitely that because the labyrinth went through this phase, it was definitely and forever a deaf ear. I don't think any function is destroyed as completely as that, but we have had gaps in hearing with the other ear excluded; and the ordinary tests would declare that the labyrinth was dead with the other thrown or to certain definite tones. I believe the labyrinth may be only temporarily out of function. I think possibly we went too far with the pendulum when labyrinthitis was discussed in 1911, and that we have too many preconceived ideas about it.

DR. H. B. BLACKWELL: I am unable to say whether or not the turning test was made prior to operation, but I do know that there was no reaction to a caloric test and with the noise apparatus in the good ear there was no hearing in the affected ear. The labyrinth in the affected ear was apparently dead, both acoustically and statically, for some time prior to the jugular excision. Richards was among the first to note an apparent association between chronic suppurative labyrinthitis and lateral sinus thrombosis. As to the return of hearing in this patient, it is a surprising fact that sometimes a patient will regain their hearing after having undergone what is apparently an attack of labyrinthitis. I have had the following experience: A few years ago within a period of a few months I was called upon to operate in two instances upon patients with labyrinthitis; the history briefly is as follows: The first patient, a young girl, was sent in to the hospital to undergo a modified radical for chronic otorrhea; routine examination prior to operation revealed the fact that the labyrinth in the affected ear was statically and acoustically deaf. Accordingly, I performed a radical operation instead of a modified radical and discovered that the cap of the horizontal semicircular canal was missing, revealing a large fistula leading into the labyrinth. I did not operate upon the labyrinth. Subsequently the patient made an excellent recovery and several months later I was surprised to learn that she could hear low voice in the ear which had been operated upon and, in addition, upon irrigating the ear with cold water, dizziness promptly ensued. Prior to operation the same ear was absolutely negative to both of these tests. Case 2 was that of a young boy about the same age, suffering with acute mastoiditis, upon whom I had performed a simple mastoid operation. Prior to the operation it was difficult for him to walk without falling to the opposite side and there was nystagmus present, with nausea and vomiting. There was nothing abnormal discovered about his labyrinth at the time of operation. Three days afterward I found that with a noise apparatus in the good ear he was unable to hear the loudest shout in his affected ear. The patient was compelled to remain in bed for the following four weeks, with all of the symptoms of an acute labyrinthine invasion. Gradually the symptoms of disturbed equilibration subsided, nausea and nystagmus disappeared and the patient was discharged from the hospital. Eight weeks after the operation I was surprised to discover that with a noise appa-

ratus in the normal ear the patient could hear a low voice in the operated ear, also that there was a caloric reaction present. A month later the hearing had returned in the affected ear to a point where it was normal.

In both of these cases the labyrinth having lost its static and acoustic functions some month later both of these functions returned; in the chronic case partially, but in the acute case a complete restoration of function ensued.

A Contribution to the Etiology of Progressive Deafness. Preliminary Report. Dr. Mark J. Gottlieb.

(Published in full in February, 1930, issue.)

DISCUSSION.

DR. J. MARCUS: Present clinical methods of the quantitative determination of the amount of enzymatic activity are not as satisfactory as they might be since no definite proportionality exists between the velocity of the reaction and the concentration of the enzyme (with the possible exception of amylopsin, in which the fact of reversability is negligible). One is often unable to obtain samples of duodenal contents containing like fractions of pancreatic secretion. The length of time elapsing between the collection of the specimen and the determination makes a difference in the results obtained. Variations in temperature at which the contents are kept during this interval (between collection and determination) will give varying results.

In spite of these often times unavoidable factors, which are bound to cause variations, one is struck by the persistent regularity of low figures (suggesting impaired pancreatic function) in the series of Dr. Gottlieb's cases.

DR. S. J. KOPETZKY: It is impossible to discuss this paper; it is in the nature of an investigation; the investigator, we all know. He reports his findings on the conditions and the premises he has established in good faith. Until such investigations are completed and until the results are checked up, we cannot discuss the matter, much as we may be tempted to theorize. Summed up in two paragraphs, thus far these results mean that a certain number of cases presenting the audiometer curve of deafness showed pancreatic dysfunction, and that is all; and that some of these cases showed transitory improvement when treated for the systemic condition. The purpose of putting the matter before the Section on Otology is to put the results and the work on record. I think that all work in a new field should be encouraged, but discussion by anyone who has had no experience in the matter would be futile.

DR. E. M. JOSEPHSON: Dr. Josephson pointed out that it is a recognized fact that disorders of the glands of internal secretion contribute to the etiology of chronic progressive deafness, though probably indirectly by their effects upon vascularity and lymphatic circulation of the inner ear more frequently than by direct effect upon its innervation. On the other hand, it is also known that diseases and pathologic disturbances may coexist with chronic progressive deafness. In cases which Dr. Gottlieb presented, of chronic progressive deafness with diabetes (high blood sugar and sugar in urine), which cleared up under insulin therapy, responses to therapy indicate definite etiologic relationship. Experience of ophthalmology with insulin therapy indicates the mechanism by which the changes in hearing may be affected as a result of insulin therapy in those cases. There have been many reports of hemorrhage in the retina, choroid, and sometimes the vitreous, following the administration of insulin in diabetic cases. These cases, coupled with the experimental data of alteration of intraocular tension upon intravenous administration of glucose solutions, indicate the probability that changes in the intravenous glucose content does affect, whether it be by altered osmotic or secretory balance, lymphatic circulation and pressure in closed lymphatic spaces. The endolymph and perilymph of the inner ear are similar lymphatic spaces, and it is possible and probable that alterations of blood sugar content have a similar effect upon the ear.

In interpreting improvement of hearing on the basis of hearing for the spoken voice, as compared with evaluation of loss of hearing determined by audiometric testing, the results cannot be considered of unquestioned value because of a tendency on the part of the examiner to alter the volume of his voice. I have found that testing of hearing by vocalization of labials, viz.,

b, d, g, p, v and z, to be more sensitive and reliable as an index of loss of hearing than either the vocalization of words or even the audiometric tests. The power of differentiation of these consonants is lost early in deafness and is, in my experience, the slowest in recovery. An additional factor, which must be taken into consideration in reporting findings as to hearing in these cases, is the variation of hearing with change in barometric pressure and temperature. It is quite characteristic of chronic progressive deafness that the patient complains to the physicians that both hearing and tinnitus are aggravated in rainy weather, when barometric pressure is lower, possibly not over half an inch of mercury, than in fair weather; or when the weather grows colder. The variation in hearing due to these causes may be so marked as to compel their being taken into consideration in the reporting and interpretation of changes in hearing, such as those mentioned in some of the cases this evening.

In view of the variability of hearing in chronic progressive deafness it is difficult to concede the differentiation of nerve deafness, otosclerotic deafness and the other types which are classed into the category of c.p.d. It is difficult to conceive of rapid variations in the pathologic structure of nerve or bone, as would account for the rapid changes in hearing observed in the disease. Changes in the external ear, the phenomenon of changes in hearing and tinnitus induced by pressure upon the carotid arteries in these cases, as well as the effects of vasodilators and vasoconstrictors, all indicate that the pathologic basis of the disease is vascular.

That there are concomitant disease processes in other parts of the body, associated with chronic progressive deafness, as well as changes more directly associated with the disease, is coming to be conceded. Pathologically it has been demonstrated that there are sometimes changes in the nerve tracts and in the hearing brain center. Clinically it can be found in many advanced cases of c.p.d. that prolonged pressure upon the carotid arteries induces epileptiform convulsions. Diminution of vision on the side of the most seriously affected ear should lead one to suspect cerebral changes.

Were there absolutely no question as to the dependability of the specified tests for pancreatic enzyme, used in Dr. Gottlieb's work, or their reliability as an index of pancreatic function, it would still be doubtful that there is an etiologic relationship between them and c.p.d., other than that both disturbances may be due to the same causes.

It would be interesting to know what were the Wassermann tests and the venereal and other histories of the cases presented.

In all fairness to Dr. Gottlieb, it should be said that he has presented his interesting material in a frank and unbiased manner.

DR. GOTTLIEB, closing: A Wassermann test was done on every case presented, and was found to be negative. A column in each chart is devoted to the concomitant conditions from which each patient is suffering.

Allergy to Argyrol in a Patient with Chronic Purulent Otitis Media. Dr. Robt. C. Howard.

DISCUSSION.

DR. L. K. MCCAFFERTY: There is very little to say after hearing Dr. Howard's excellent report. I saw the case with Dr. Howard, and it impressed me as rather unique as to sensitization from argyrol. In the skin clinic we see reactions to all sorts of substances, but I do not recall seeing any from argyrol or other silver preparations. It was applied in the nose, the patient absorbed it, and it reappeared on the face; apparently being conveyed through the bloodstream, rather than in direct contact, although I believe that some argyrol did come in contact with the skin and produced a dermatitis. The patient also had urticaria and some edema over the body. It is certainly a case well worth reporting in the literature and I believe that if we follow up some of our skin cases more carefully we may find similar ones. I am very glad to have had the opportunity to see this patient and hope that Dr. Howard will allow us to show the case at the next meeting of the Dermatological Section, for I am sure the members would be glad to see it.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Tuesday, Jan. 7, 1930.

A Study of Mastoiditis; a Resume of 644 Cases. Dr. George M. Coates, Dr. Matthew S. Ersner and Dr. Abram H. Persky.

(Published in full in April, 1930, issue.)

DISCUSSION.

DR. COATES: The credit for this paper belongs to Dr. Persky, who spent a good deal of time going over the records of the Medico-Chi and Graduate Hospitals to get the mass of data that he has collected. You will note, of course, that the series, although a respectable one, is not large, but it must be remembered that it represents only one otological service in one hospital, since at the time the Medico-Chi was closed, we were transferred to the Graduate. I think the number of cases operated upon will bear evidence that our service has been conservative, since some cases recovered without operation, and many were kept in the hospital for considerable periods of time before operation was resorted to. In spite of this and when we bear in mind that many of the cases admitted were in reality advanced cases, our mortality cannot be called particularly high.

The report on the bacteriological side is not a good one, for on our records there are but a comparatively few bacteriological reports. A few of the cases were undoubtedly sterile, but owing to the difficulties of getting the specimens taken at operation from the Medico-Chi Hospital across town to the laboratory situated in the Polyclinic Hospital, many specimens were dried up and failed to produce growths. These difficulties, bad enough at any time, were increased when operations were done at odd hours, such as late at night. It is my belief, however, that in every case that was operated upon, the culture was taken.

As to the X-ray findings, after close co-operation was established with the radiological department, splendid results were achieved. At first we found discrepancies between the reports on the films taken of our mastoid cases and the findings at operation. We then established a rule that the otological department should first go over the films with the radiologist and that afterwards, wherever possible, the radiologist should come to the operating room and see for himself the operating findings, after which a restudy of the plates was made by both services. In this way, in a very short time, the radiological laboratory was not only making beautiful pictures, but gave us very correct and helpful readings.

We had a few cases of mastoiditis develop after a submucous resection had been done, evidently due to the nasal packing. In my experience, when acute middle ear suppuration develops from this cause, it is not unusual to have the suppuration extending into the mastoid. There were also four cases of mastoiditis occurring after a tonsil and adenoid operation, which we felt was probably due to the practice prevalent at that time of scrubbing out the nasopharynx with gauze. Since that practice has been eliminated, we have not had any trouble from this source.

In many of our cases we used some type of blood clot closeure and our successes by this method were reasonably numerous. When it failed we do not feel that the failure was due to any particular type of organism, but probably to an increased virulence, due to a prevailing epidemic or to the poor resistance of the individual. In 1917, in one army camp in the South, there were many mastoid cases in soldiers, who were very poor physical specimens, hookworm carriers and who contracted in camp any or all of the ordinary diseases of

childhood, such as measles, mumps, scarlet fever, etc. We speedily found that those individuals who had developed no resistance were very poor subjects on which to try a blood clot dressing. Our failures were universal. Later, in another camp, where there was a body of men in excellent physical condition, there were few or no failures by this method.

DR. ERSNER: It is gratifying to participate in the discussion of Dr. Persky's paper. It concerns the work that Dr. Coates and I have been carrying on for so many years. It brings back a great deal of joy, but when I learned from the statistics that 29 deaths occurred, although a small percentage, it rather shocked me.

It was reported in this group that measles was the "bugbear" in the causation of mastoid disease. My impressions, as well as statistics that were gathered from other sources, are that scarlet fever is the greater factor of the two that is responsible for acute mastoiditis. The variation in the causation of mastoiditis is probably due to the fact that very often when a scarlet fever case is diagnosed, it is immediately shipped to a hospital for contagious diseases, and we lose track of it. On the other hand, most measle cases are generally treated at home, and when an acute mastoid develops, the acute measle symptoms have already subsided and the quarantine period is over. The patient is then admitted into a general hospital for observation for mastoiditis. This probably explains why our statistics show measles as the foremost causative factor in mastoiditis. It will be interesting to watch the future statistics of acute mastoids and their relationship to scarlet fever. With the work that is being accomplished by the Dick's there is no question that scarlet fever will be eliminated just as well as diphtheria, typhoid fever, etc. Naturally the percentage of mastoids that develop following scarlet fever will drop.

The facilities for carrying on our bacteriological studies were very much handicapped as the infected material had to be transferred from the Medico-Chirurgical to the Polyclinic Hospital Laboratory. Very often many of the cultures were contaminated and a great many of the swabs were dry before they reached the laboratory.

In a group of 100 mastoids, where a special technician was engaged, we found that 65 per cent of all organisms isolated were those of the streptococcus hemolyticus.

Mention was made that swimming did not play an important factor in the causation of mastoiditis. This can be explained by the fact that Dr. Coates and the most active participants in his clinic are vacationing during that period and we therefore only had a few isolated cases that we could refer to.

It was inferred that a case of secondary mastoiditis developed following toxin antitoxin administration. I believe that this were merely a coincidence. Occasionally we administer antitoxin as a nonspecific proteid where the healing of mastoid wounds is unduly prolonged. Since toxin antitoxin is being employed as a general routine in preventaive medicine, we must abstain from promiscuous administration of antitoxin as often a severe anaphylactic shock results.

It is interesting to note the table illustrating the time element, showing the dates from the onset of the disease to the date of operation. Our minimum was 12 days and the maximum was three to four months before operation took place.

Mastoid infections may be either epidemic or endemic and they usually occur on the heels of acute infection and run in waves. For instance, in 1928, there was an early fall and an early spring run of mastoids. In 1927, most of the mastoids occurred about midwinter, and this year there have been very few.

In my experience, X-ray is an indispensable factor and I utilize it in all mastoid conditions as an aid in diagnosis.

The symptom of pain was referred to but not sufficient stress was placed upon its occurrence and character. Subjective pain occurring at night and

pain that does not respond to sedatives should be carefully guarded as it is very often pathognomonic of mastoiditis.

The question of performing simple mastoidectomies in order to save hearing is just as important as operating to save life. In early mastoid disease the obstructive element is the most pronounced. As the disease progresses and nerve deafness begin to appear, it is time to consider mastoid operation in order to save the patient's hearing.

It was inferred that mastoid infection may spread by contact. This can be substantiated by the fact that mastoid operations were performed on individuals of the same family, at the same time, especially in twin groups.

It is true that submucous resections may be complicated by acute mastoids. This may be due to excessive packing, indiscriminated nasal irrigation and an acute infection occurring in the upper respiratory system.

DR. MACKENZIE: A paper of this kind is hardly debatable, but a few thoughts occurred to me. I was interested to learn that the submucous resection was responsible for mastoiditis in some of your cases. I had the privilege of observing two such cases; both had diabetes. One was seen in Politzer's Clinic, having been operated one week before in Chiari's Clinc; and the other was a duplicate case seen in one of the Philadelphia clinics.

The blood count as offered in the paper of Dr. Persky is typical and checks with my own experience in mastoid disease, with its variations in complications.

The essayist referred to the fact that in all the cases where the symptoms were 100 per cent for mastoid disease, at the time of operation he found there was justification for the operation. I am reminded of the fact that in my experience, where a patient has but 30 per cent symptoms and signs of mastoiditis, in practically 100 per cent of the cases at operation we find the diagnosis confirmed.

I have no complaint to make against the X-ray man's interpretation of the films made of mastoids. I think it is largely a matter of the selection of the X-ray expert. The man to whom I send most of my cases for X-ray diagnosis has approached 100 per cent accuracy. This includes some of the cases which I have presented before this Society, in which there was a locked-up carcinoma in one, necrotic bone without frank symptoms of mastoiditis, and other rather rare conditions.

As for the cases in which the patient gives the history of a previous discharge with a healing of the drumhead and the absence of frank symptoms of mastoiditis, there is frequently a latent mastoiditis present that requires all the skill of the otologist and roentgenologist combined to make a diagnosis.

I do not recall any mention having been made of vertigo or nystagmus in the series of cases reported this evening.

Will Dr. Persky be kind enough to tell us what happened in the cases that were not operated? What was the character of the conservative treatment and the result? Furthermore, what happened to the hearing function? As to the complications that were present and referred to in the paper, I would like to ask were they present before or after operation.

A mortality of only 4 per cent, including the cases with complications, is a result that speaks for itself.

Dr. Coates and his entire staff, including Dr. Persky, should be congratulated for the excellent presentation we have listened to this evening.

DR. WIEDER: I was particularly interested to note that Dr. Persky called attention to the possibility of infection by contact. About six-and-one-half years ago, I reported two cases of mastoiditis, one of which developed meningitis, and the playmate of this child promptly afterward developed otitis media, mastoiditis, meningisms and labyrinthine irritation. Prompt operation saved that child. She had 5,000 leukocytes in the spinal fluid, but no bacteria. At

that time I called attention to the danger of a healthy child playing with another child while in the acute stage of otitis media and the possibility of contracting infection. I also pointed out the danger from school children that go back to school too early after an infection, spreading the infection by contact of infected hands with pencils, pens, slates, etc., used by other children.

DR. ALEXANDER: There is no doubt but that in the last two or three years the Roentgenologist has developed a substantially improved technique, with results that I am sure must be of more than ordinary interest to all, and to the aurist surgeon in particular, as up to about three years ago a Roentgenograph of the mastoid region furnished little information of a mastoid condition that was of value or help from a diagnostic standpoint.

Some months ago I was consulted by two physicians, one an otologist and the other a surgeon, on a certain case to help decide the presence or absence of a mastoid abscess. The symptoms presented were headache, an occasional chill, intermittent high temperature and an intermittent small amount of discharge from the ear. The picture on the drum membrane was rather insignificant and the patient did not at any time feel very ill. There were no major symptoms of mastoid involvement and the two physicians could not agree upon the presence of a mastoid condition. After making a careful examination of the case I was convinced of the presence of mastoid disease. We then consulted an X-ray picture of the involved mastoid process that demonstrated beautifully the presence of mastoid pathology. The other otologist, however, remained unconvinced.

Upon removing the external cortical plate of the mastoid process, "which was diploetic in type," we found well established congestion of the bony walls and linings of the small cells in the body of the process and pus in the larger tip cells.

I mention this case in particular, inasmuch as it seems to be one of peculiar value as an example of the advancement and accuracy in the technique and skill of the rhinologist, because of the type of mastoid process and the pathology found therein.

DR. STOUT: When you send a specimen of purulent material taken from the mastoid area while performing a mastoidectomy in acute cases and the report comes back with findings of no bacteria, I would suggest that more material be taken either from the field of operation or from the external canal, if there is a perforation in the drumhead, and send the second specimen with a request to examine for anaerobic organisms. These organisms are sometimes found in places where least suspected.

DR. STAUFFER: I have been doing T. and A. operations for 20 years and have never had a mastoid case following a T. and A operation. I do not know why it is that certain hospitals have so many more complications than others. I have often wondered why we have our cultures come back sterile. Some say it may be due to the fact that they are dried.

I am inclined to agree with the essayist that temperature after operation is as important as temperature before operation.

DR. PERSKY, in closing: There is a very little more that I have to add. Dr. Mackenzie brought up several points; the first is that of nystagmus. This question is an open one, for the cases were usually in the hospital so short a time prior to operation that we did not have an opportunity to study this phenomenon. However, wherever it was conspicuous we have noted it. In the above report I have noted three cases of positive fistula test.

The question of the presence of a brain abscess in the above series of cases has been brought up. While some of these cases with this complication came in prior to operation and a diagnosis was so made, on the other hand, the other group of cases of brain abscess came in the hospital only after a preliminary mastoidectomy had been performed. I am unable from these above statistics to differentiate the two groups.

I have a list of 35 cases that did not have any operations. They were admitted for observation and were studied for their response to local treatment. Some of these cleared up under this local treatment and were discharged as cured, while others refused operation and what was the ultimate result of these cases, I am unable to determine.

We are not in any position to determine what the postoperative result was on the hearing of our patients. The majority of these patients were from the private services and the hospital lost track of them.

The Role of the Eosinophile in Certain Otological Conditions. Dr. Charles C. W. Judd.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ERSNER: Dr. Judd's paper brings us a message. His citation of the lytic element and its relationship to the eosinophile should be carefully studied. Several years ago, Dr. Coates and I started a study of cases on maxillary sinusitis and its relationship to allergy and asthma. We observed that the eosinophile was the predominant cell found in the mucous membrane obtained from the maxillary sinus of patients suffering from asthma. We ascribed these findings to protein disintegration and enzyme change which, basically, probably are lytic. The eosinophile was also found in many allergic phenomena, as well as in nasal congestions caused by nonbacterial irritations. Our observations coincide with Dr. Judd's; that is, that the eosinophile disappeared from the tissues where acute infection occured. The eosinophile increases to which Dr. Judd referred are probably due to the lytic phenomena or to the stimulation of the leucohematopoietic system of which the eosinophile is a member. When bacterial infection becomes more pronounced there is a disproportionate increase in the polymorphonuclear leukocytes and therefore the proportionate diminution in the eosinophile. The metamyelocyte described by Schilling and Arneth should also be watched carefully as these cells may be utilized as an indicator in mastoid disease. It will be interesting to observe whether there is an increase in the eosinophile following a mastoid operation. We have all noted the postoperative reaction, which is characterized by rise in temperature following mastoid operation. These rises are probably due to autogenic blood disturbances, which probably causes secondary lytic changes. In the olden days the surgeon called it fibron absorption and today, according to Judd, we may call it autogenic lytic phenomena.

In conclusion, I wish to congratulate Dr. Judd for the message that he brought to us.

DR. JUDD, in closing: Though fully aware of the significance of the Arneth count, not only as a general means of ascertaining the degree of bone marrow activity, but also as applied to the conditions under consideration, I purposely avoided any mention of this feature because emphasis has already been placed upon it. On the other hand, the eosinophile cell which is so easily seen is practically lost sight of. I just wanted to bring to your minds that the eosinophilic cell is not only good to look at, but is likewise important.